Hospital Library

JAN 15 1936

VOLUME 32 NUMBER 1

ARCHIVES OF SURGERY

EDITORIAL BOARD

DEAN LEWIS, Baltimore

EVARTS A. GRAHAM, St. Louis WALLACE I. TERRY, San Francisco

ALTON OCHSNER, New Orleans WILLIAM DARRACH, New York

EDWARD STARR JUDD, Rochester, Minn.

JANUARY 1936

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, July 10, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized July 29, 1920.

NOTICE: 1936 SUBSCRIPTIONS NOW DUE

Subscriptions for the current year are now due and payable, if not already paid. An envelop-remittance blank, goldenrod in color, will be found in this issue. For the convenience of those who subscribe for more than one, all of the periodicals published by the American Medical Association are listed. A check mark may be placed before the names of the periodicals desired, or the names may be written on the lines provided in the blank. Thus, one check or draft may be sent to cover the entire amount of the subscriptions.

COPYRIGHT, 1936, BY THE AMERICAN MEDICAL ASSOCIATION

ARCHIVES OF SURGERY

VOLUME 32 JANUARY 1936 NUMBER 1

SKELETAL CHANGES IN MALUM PERFORANS PEDIS

JACOB KULOWSKI, M.D.
ST. JOSEPH, MO.
AND
ROBERT PERLMAN, M.D.
BROOKLYN

The fact that osteomyelitis or a similar change in the bones is a factor of great importance as the underlying local pathologic process in perforating ulcer of the foot is insufficiently appreciated. This study is based on observations on 33 cases of malum perforans pedis. Perforating ulcer of the foot was first described by Vesigne in 1852, and the condition was designated by Leplat in 1855. The underlying changes in the bones are significant in explaining some of the basic factors of the condition. The skeletal changes in this condition were noted and described by Savory and Butlin as early as 1879 in a paper which remains a classic. Bony sequestration formed a prominent feature of the clinical picture in their 5 cases. They stated that perforating ulcer might well begin in the depths of the foot or that the initial processes might begin almost simultaneously throughout the whole depth of the sole from the skin to the bone. This remarkable observation still forms the basis of any discussion of the pathogenesis of the disease today. Scaglietti in 1931 concluded that such pathologic change in the bones is secondary to the ulceration of the skin but is also in part independent of this, as is shown by the early primary trophic changes in the bone even when the cutaneous areas are not involved. He also expressed the opinion that the vascular changes are secondary to the cutaneous infection and are not primary, as the French believe. Finally, the changes in the bones must be considered coordinate with the cutaneous changes, as there is no proof that the skeletal changes are the cause of malum perforans pedis.

A brief historical review revealed that all the known major etiologic factors in the disease, such as predisposition, familial tendency, trauma, arterial disease, disease and influences of the peripheral nerves and the central nervous system, pressure and constitutional diseases, such as diabetes and leprosy, were considered major factors between the years 1852 and 1873 and even earlier. The neuropathologic theory has steadily gained ground since then, although there is still some difference

From the department of orthopedic surgery, State University of Iowa, service of Dr. A. Steindler.

of opinion as to the exact nature of the lesions. This theory is also supported by the fact that in even the mild varieties of leprosy, which has a remarkable affinity for the peripheral nerves, there are marked changes in the bones, and in many instances the nervous lesions predominate, although many of the changes are unquestionably due to bacterial invasion.

Defective innervation may favor the occurrence of ulceration in two ways: first, by leading to a break in the continuity of the surface, thus permitting the introduction of micro-organisms, and, second, by depressing nutritional activity and subsequently diminishing reactive resistance to the bacterial invasion. The skin and subcutaneous tissues are richly supplied with sensory nerves. Disordered nerve function may lead to profound changes in the tissues, for example, acute bed sores in a case of transverse myelitis within a few hours of the onset. Occasionally a lesion may appear or occur as a pure plantar lesion apart from any such influence as an incidence of trauma, suppurating wart or corn or even chronic epithelioma. All the etiologic factors may emerge as of primary or secondary significance in the pathogenesis. There is apparently a familial tendency, for the condition begins in many cases in early adolescence, as was indicated in our series by a study of the incidence according to age in decades. There were 2 cases in the first decade, 4 in the second, 3 in the third, 6 in the fourth, 8 in the fifth, 5 in the sixth, 4 in the seventh and 1 in the eighth.

It was evident from a study of our group that more than one factor was responsible for the disease, especially in case of such a basic disease as tabes dorsalis. This was illustrated in a man aged 45 whose foot was crushed sixteen years previously. Six years later there developed a typical ulcer. Subsequent periodic examinations revealed tabes dorsalis.

In this series the following etiologic factors were noted: diabetes in 6 cases, obesity in 1, vascular changes in 8, trauma in 4, encephalitis in 1, tabes dorsalis in 2, a familial tendency in 1, spina bifida occulta in 5, spina bifida manifesta in 1, traumatic myelitis in 1, a roentgen ray burn in 1 and frost-bite in 6. It is apparent that essentially there must be several basic factors, such as constitutional predisposition, trauma, constitutional diseases and vascular and nervous influences. Two main divisions would be sufficient for all clinical purposes: the idiopathic and the symptomatic. The term malum perforans pedis must therefore be interpreted as a generic term in order to clear up the present clinical confusion.

The variable known and unknown factors in the disease are readily appreciated from a brief consideration of the pathogenesis in some of the cases under consideration. In the 6 cases in which there was a history of freezing, ulcers formed on each foot five years later. Unilateral ulceration appeared fifty years after freezing in another case. In 1 other case of the same duration ulceration of both feet resulted. Of

57 cases of spina bifida manifesta entered in the records of the hospital we know of only 3 in which plantar ulceration resulted. Of course most of the patients were probably too young and had not yet subjected their feet to sufficient functional traumatization. Of 136 cases of spina bifida occulta with symptoms of sciatica, residual ulceration developed in only 1 and that only after the development of postdiphtheritic paralysis. It is generally conceded that weight-bearing and pressure effects are extremely important, as borne out by the fact that in some of the cases the ulcer healed while the patient was receiving treatment, which consisted chiefly of rest in bed, and as promptly reappeared after the beginning of weight-bearing.

This was strikingly exemplified in a man aged 59 years, who walked on the outer border of the foot following malunion of a compound fracture of the leg, with the formation of an ulcer on the outer border of the foot one year later. A study of the relative sites of ulcers also emphasized the significance of pressure effects.

The area of the joint of the big toe was involved in 14 cases, the region of the joint of the fifth toe in 12, the heel in 6, the lateral border of the foot in 3, the rest of the anterior portion of the arch in 10 and the dorsum of the foot in the 2 cases in which clawing was noted. In 7 cases lesions were present on both feet; in 3 there was more than one ulcer on the same foot. It is of interest to note the following associated anatomic abnormalities: hammer toe in 1 case, clubfoot in 4, clawfoot in 2, bunion in 1, spina bifida occulta in 5 and spina bifida manifesta in 1.

The condition predominates in men, chiefly because of occupations which necessitate added strain and traumatic hazards and probably because men are more liable to nervous and vascular disease, which seem to be important in the etiology of perforating ulcer. Of the 33 patients, 24 were males. The average age was 43 years, and the ages ranged from 13 to 77 years. The average age for the females was 34 years, and the ages ranged from 4 to 61 years.

Some authors have claimed that pain is contrary to the diagnosis of perforating ulcer. In this series there were 11 cases in which there was a definite history of pain (perhaps due to associated vascular disturbances), and in 10 the history was questionable. Of these, there was moderate intermittent discomfort in 5. Of this group there were 2 cases of the idiopathic type, and in the rest the condition was symptomatic. In the latter group there were 2 cases of diabetes, 2 of spina bifida, 2 with a history of freezing, 1 of tabes and 1 in which there was definite clinical evidence of vascular disturbance of the extremities. Pain as a symptom may therefore be present in degree and is dependent on the underlying general etiology and the extensiveness of the local pathologic changes. There is generally a localized area of anesthesia immediately surrounding the ulcer. There is usually very little tenderness on pressure, although walking may cause symptoms. The foot sooner or later

becomes the seat of other symptoms pointing toward impairment of the nerves or vessels such as increased growth of hair, hyperhidrosis (even bromidrosis), pigmentation and alteration of the nails.

Adrian described the development of the ulcer as follows:

1. A callus forms at one of the characteristic sites of predilection, with thinning of the epidermis and the formation of a blister. The contents are ordinarily serous but may be bloody after trauma. After this perforates or ruptures there is the usual formation of the ulcer.

2. The ulcer is obdurate and does not heal or may illustrate the characteristic tendency to periodic recurrent healing and reformation.

3. A residual deep ulcer forms, associated with tenderness and involvements of the bursae, bones and joints. Suppuration is now the rule, and the infection may burrow deeply and affect the tarsus even before it reaches the surface, because of the calloused soft structures. After partial destruction and elimination of bone occur, there follows shortening of the entire foot, as may occur strikingly in leprosy. Erysipelas, lymphangitis, abscess or cellulitis is frequently observed.

Scaglietti summarized the histologic changes as follows:

1. In the skin there are an increase in the size of the sweat glands and hyperkeratosis. The base of the ulcer may have granulations, or the floor is covered or formed by dense connective tissue.

2. Changes in the bone occur chiefly in the metatarsophalangeal joints. Even when the joints are not involved by the cutaneous ulceration, the cartilage of the joints shows superficial necrosis. There are pannus-like formations and resorption of cartilage from the superficial and subchondral zone. If the joint is invaded, fibrous ankylosis or dorsal dislocation of the first phalanx is a minor consequence. The ends of the joint and part of the diaphysis may be destroyed. The ends of the diaphysis show concentric atrophy and pointed ends. In the metatarsal bones osteomyelitic changes usually follow infection of the joint, and all the following changes may be observed: necrosis of the suppurated portion of the bone, maximal porosis of the intact parts, formation of a subperiosteal abscess, periosteal osteophytes, sequestrums with granulation tissue and formation of a scar and, finally, healing after elimination of the sequestrums. The abscess cavity of the bone fills with new spongy bone, with inclusion of old dead bone in the new spongy trabeculae.

Scaglietti's studies also demonstrated the following histologic skeletal changes in three stages:

1. Between the lacunar remaining parts of the old bone, new fibrous formations of bone, with islands of cartilage, appear in fibrous bone marrow.

2. Tiny new bone trabeculae become denser and more numerous and finally more and more mature until they are lamellar.

3. Bone becomes transformed and substituted by purely lamellar bone, in which trabeculae of small parts of the primitive bone and cartilage may be included as remnants of the first and second periods. The vessels show marked thickening of the intima, with narrowing of the lumen or thrombosis in different stages of organization.

The results of our studies bear out these observations and also emphasize the extensive collateral changes and tremendous regenerative efforts of the elements of the bones and joints, in spite of the intensive destruction present and the apparent hopelessness of the disease clinically. Our therapeutic experience and that reported in the literature was most discouraging so far as clearing up the local condition was concerned. Prior to their admission to the hospital 8 patients underwent some type of surgical procedure: drainage and sequestrectomy were performed in 2 instances, excision of the ulcer in 1 and amputation in 9 (7 of toes, and 2 of a foot). In the clinic, drainage was done in 12 cases, excision of the ulcer in 2, sequestrectomy in 3 and amputation in 12 (in 8 of toes and in 3 of a foot in toto, with amputation through the transmetatarsal joint in 1). Of this entire group healing occurred in only 2, and in 4 there was improvement.

It has been firmly established that an indirect method of attack directed toward improvement of the circulation must be utilized. Surgical procedures were developed and centered about the sympathetic nervous system following the observation of Claude Bernard in 1851. These procedures ranged from direct attack on the chains and ganglions of the central sympathetic system to the periarterial decortication of Leriche. McClintic in 1929 recommended periarticular injection of alcohol instead, because the effect is immediate and less formidable. It is known today that effects obtained from a surgical procedure on the sympathetic nervous system are not permanent. All cases in which this type of treatment is to be used must be carefully selected after a thorough investigation as to the presence of vasospasm and vascular response following paralysis of the vasomotor fibers. Such dermathermic studies were made in several of our cases with little or no response, as attested by a definite rise in the surface temperature.

Because in so many cases of the disease there are primary or secondary vascular changes, it would appear that the pavaex method of vascular exercise is indicated either as a prophylactic or as a primary or adjuvant therapeutic measure. The amount of local surgical treatment is determined by the amount and degree of change in the soft tissue and bone, changes in the bone being also largely secondary to ischemia. Although we have not had an opportunity to employ the pavaex method, we strongly recommend its trial and make this suggestion in the hope that others will give it their favorable attention. It should do much to minimize the necessity for amputation.

REPORT OF CASES

Because detailed histologic studies on amputated limbs affected by malum perforans pedis have not appeared in the English literature, the following analysis and study of a case is given.

CASE 1 (figs. 1 and 2).—*History and Course.*—Ruth H., aged 8 years, was admitted to the hospital on August 1923. A varus deformity of the right foot had been noted since the age of 2 or 3 months, and progressive curvature of

Fig. 1 (Ruth H.; case 1).—Photographs and roentgenograms of the condition diagnosed as chronic osteomyelitis with complete transformation of the bony structures, showing extensive destruction and characteristic osseous absorption.

the spine had been present since the age of 2 or 3 years. The patient walked at 14 months. Examination revealed some trophic changes: the right foot shorter than the left by ¾ inch (1.9 cm.), slight varus, slight malformation of the nails and slight cyanosis. The flexor muscles of the toes and the peroneus muscles were weak, presumably a congenital weakness due to spinal malformation. The right knee jerk was sluggish.

An operation for spinal fusion was performed in 1926, followed by an uneventful recovery. Ulceration on the ball of the big toe appeared in 1928. It was

cleaned surgically and closed, but the wound never healed. The pathologic report stated that the diagnosis was chronic bursitis.

In 1933 there were three ulcers on the anterior part of the arch, with slight purulent drainage. In July drainage by the Orr method was instituted, and sequestrectomy of the first and fifth metatarsal bones was performed. There was considerable drainage one month later. In September a large area of devitalized skin appeared at the base of the big toe (fig. 1). On September 15 the remainder of the fifth metatarsal bone was removed, and the wound was cleaned. The wound was closed tight, the cast was applied, and the wound healed. In October the wound had remained healed.

Laboratory examination gave negative results, and the dermathermic reading was unsatisfactory. Roentgenograms showed extensive destruction and charac-

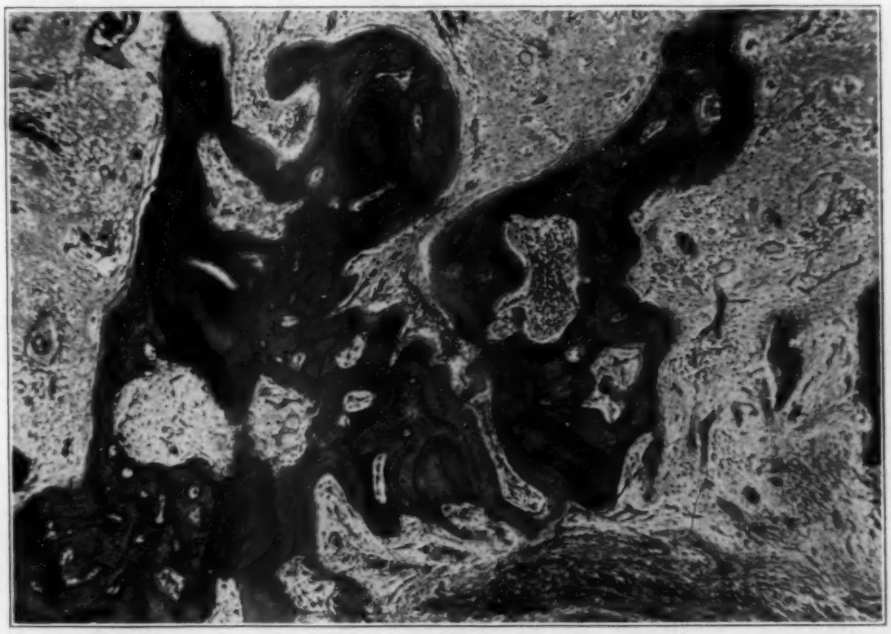


Fig. 2 (Ruth H.; case 1).—Photomicrograph of one section of the fifth metatarsal bone, showing radical pathologic changes.

teristic osseous absorption, chiefly in the metatarsophalangeal joints. The other bones were also extensively involved, and sequestration was present.

In 1934 amputation through the lower third of the leg was performed owing to the recurrence of ulceration and drainage. The stump healed.

Examination of Metatarsal Bone.—The bone was very deformed. It was markedly thickened and somewhat shortened. The distal end was covered with joint cartilage which was thinned out and showed erosion in some places. The surface of the bone was irregular. The bone was cut longitudinally, and it was found to consist of rather dense spongy bone of normal hardness and of hyperemic fibrous bone marrow. There were no signs of suppuration or of necrosis.

Two slides were prepared for histologic examination. Both sections showed essentially the same picture (fig. 2). There was a rather dense spongy bone with

very irregular structure. The bony trabeculae showed many blue cement lines, most of them typical lines of apposition, as signs of periodic apposition of bone. Some of the bony trabeculae were united by a rather young bone tissue of callous character, and the irregularity of the structure of the single bony trabeculae combined with callus formation between the bony trabeculae brought about the rather complex and irregular structure of the entire spongy bone. The bone marrow was fibrous, with many large capillary vessels. A considerable amount of chronic fibers of the fibrous bone marrow were separated from each other by edema and in other places by hyaline degeneration and by deposition of osteoid tissue. The cartilage of the joint was markedly thinned out by resorption from the cavity of the joint, with loss of the normal number of basophils but no necrosis of the cartilaginous cells. The peripheral portions of the cartilage showed complete resorption by a pannus-like formation from the synovial layers. There was not, however, a pannus-like formation of connective tissue on the surface of the cartilage itself. The synovia showed marked infiltration by lymphocytes and to a great extent by plasma cells. In one area close to the proximal end of the bone the marrow spaces were open, and the surface of the bone was covered with nonspecific granulation tissue. This granulation tissue blended with its deeper layers continuously into the fibrous bone marrow. The diagnosis was chronic osteomyelitis of the fifth metatarsal bone with complete transformation of the bony structure.

Examination of Amputated Leg.—Dr. Ernst Freund made the following analysis: The leg was amputated through the middle third, and formaldehyde was immediately injected into it. The foot was markedly deformed, especially by being shortened in the anterior half and by the dorsally subluxated contracted toes. The big toe overlapped the second owing to an extreme hallux valgus. The toe-nails showed signs of onychogryposis, with almost complete atrophy of the fourth and fifth toes. The skin on the dorsum of the foot exhibited hyperpigmentation. There was no thenar or hypothenar eminence on the plantar aspect, and the anterior portion was bulbous, densely calloused and scarred, but there was no evidence of ulceration. The joints and bones of the foot were inspected and prepared for histologic studies.

There were no gross changes in the tibio-astragalar joint. However, marked degenerative arthritic changes were noted in Chopart's joint between the head of the astragalus and the scaphoid bone. The capsule was thickened and villous. The cartilage was degenerated unevenly and exhibited some marginal proliferative activity. The medial denuded area of the joint showed healing by a layer of fibrous tissue and cartilage. The anterior portion of the subastragaloid joint was similarly involved, while the posterior portion was unaffected, which leads one to conclude that no true communication exists between these portions and that this joint has no anatomic continuity. The anterior surface of the joint of the scaphoid bone did not show such marked changes as the posterior surface, but there was considerable atrophy of the cartilage, with peripheral retraction. The first metatarsal bone was destroyed in its anterior half, as was the adjoining portion of the first phalanx. The intervening joint was obliterated by a scar, which also filled a funnel-like depression fracture in this region. The second and third metatarsal bones showed destruction and were completely out of line with their respective phalanges. Dense scar tissue proceeded into these areas from the callus on the plantar surface. The fourth metatarsal bone, which exhibited in the roentgenogram (postamputation) typical pseudo-arthrosis, revealed a well outlined cavity of the joint which was situated in the midportion of the bone. As the roentgeno-

gram showed very little of the fifth toe, this portion of the foot was prepared in toto for further microscopic examination. Portions of the entire foot were sectioned and stained.

Astragaloscaphoid Joint: The cartilage of the joint showed marked degenerative changes. Old hyaline cartilage was present, but in no place was there an entirely normal appearance. The surface was very irregular; the superficial layer with a horizontal direction of the cells had disappeared, and the ground substance was fibrillated. The superficial cell groups showed signs of proliferation. Large cell capsules formed and became gradually sequestrated into the cavity of the joint. In other places, where the fibrillation of the surface of the joint was not so marked, the superficial layers of the cartilage showed partial disappearance of the hyaline ground substance, resembling fibrous more than hyaline cartilage. These changes extended far down into the pressure zone of the cartilage of the joint and were accompanied sometimes by the formation of fissures in a radial direction, with fibrinoid impregnation of the walls of the fissures. At the margins of the joints, especially at one side, typical exostoses had formed. The synovial tissue covered the thin cortex at the joint along a slightly lacunar line which frequently showed osteoclastic resorption. The zone of calcified cartilaginous tissue was at the central area of the surface of the joint and fairly well preserved. Toward the margins, however, the old zone of calcification had disappeared almost entirely. These superficial marrow spaces contained a hyperemic, loosely fibrous bone marrow. In many places osteoclasts could be seen busy with resorption of the bone and calcified cartilaginous tissue. The subchondral bone was slightly porotic and showed besides porosis, a pathologic structure. One was still able to outline easily the old thinned-out bony trabeculae running in a static way, chiefly radially, to the surface of the joint. Corresponding to the apex of the surface was a slightly osteosclerotic bony change, in the sense that between the old bony trabeculae irregular bony bridges of newly formed bony tissue appeared. This new bone was chiefly fibrous. It was also formed along the surface of the old bony trabeculae so that sometimes marrow spaces between bony trabeculae could be almost entirely filled out by new bone tissue. This picture resembled that of metastasis of osteoplastic carcinoma. It was of great interest, however, that the astragalus, which until now showed purely degenerative and not inflammatory changes of the cartilaginous and bony tissue, already showed this peculiar type of osteoporosis associated with a strong osteosclerotic factor.

Anterior Subastragaloid Joint: The changes in the subastragaloid joint were essentially the same as those described for the head of the astragalus, though maybe a little more marked. Exostoses had developed at the margin of the joint but as yet did not surpass the level of the old surface. The subchondral bone tissue was porotic, but the osteoporosis was certainly on the way to repair. Most of the bony trabeculae were of complex structure. The central portion was represented by the old porotic bony trabeculae, at the surface of which new bone had become apposed either in a straight line or more irregularly, sometimes forming bridges from one trabeculae to another. How these bridges form could be seen in one spot closer to the surface of the joint. Here were a few porotic old bony trabeculae embedded in loose fibrous bone marrow. Within the fibrous tissue and at the surface of the old bony trabeculae new spongy bone had formed irregularly, fibrous and in great part still osteoid. This formation of bone occurred primarily without the participation of osteoblasts. It was an infiltration process between the fibers of the fibrous bone marrow. One could follow all the stages of fibrous bone tissue from the first stage of infiltration, with an edematous fluid, to the more solid

stage of osteoid tissue, without sharp outline, and to the formation of bony trabeculae, with beginning calcification and surrounded by a dense layer of osteoblasts. All the changes could be considered as purely degenerative. There were no signs of inflammatory reaction. The invasion of the deeper cartilaginous layers from below in some places reached a pronounced degree. Marrow spaces had invaded the lower one half of the thickness of old cartilaginous tissue and had brought about a very irregular structure, with a mixture of old calcified and noncalcified cartilaginous, fibrous and fatty bone marrow, with osteoclasts and new bony trabeculae in the new marrow spaces. These pictures showed how the epiphysis gradually became enlarged by the recuperation of endochondral ossification at the cast of the old hyaline cartilaginous layer of the joint.

The changes at the posterior concave and anterior convex surfaces of the scaphoid bone were still typical of degenerative hypertrophic arthritis. The whole scaphoid bone showed more pronounced osteoporosis than that which could be observed in the astragalus and calcaneum. The subchondral bone tissue, however, showed the same kind of osteosclerosis, and all the stages from osteoid fibrous tissue to an almost complete obliteration of the marrow spaces by new bone tissue could be seen.

The skin of the big toe on the plantar side showed hyperkeratosis, which in some places was so marked that it could not be cut. But there were no defects in the skin caused by the formation of ulcer. It was surprising to note the great amount of sweat glands which with their hyperplastic convolutions of tubules occupied the subcutaneous fat tissue.

As far as the skeleton of the big toe was concerned, in no place could normal structures be found. The least amount of change was seen in the end phalanx. This showed considerable atrophy involving both the cortical and the spongy bone. Lacunar resorption was still going on at the outside of the cortex. The bony trabeculae of the spongy bone, although porotic, showed an abnormal amount of cement lines, which showed that the bone tissue had undergone, previous to the actual osteoporosis, changes with thickening by the apposition of bone. The bone marrow was almost pure fatty marrow, without any inflammatory signs. The joint between the proximal and the distal phalanx showed typical fibrous ankylosis, with slight subluxation of the distal phalanx. Old hyaline cartilage was present only as a very thin layer without any basophilic activity in the ground substance. The more superficial layers had disappeared by the process of incomplete cartilaginous resorption described by Weichselbaum and Pommer. The fibrous pannus-like formation on the surface was creeping over the cartilage of the joint. The cell groups, some of which were enlarged, formed the so-called lacunae. Under gradual disappearance of the ground substance between the cells, groups of the latter merged and became included in the fibrous tissue filling the joint space. The loss of hyaline cartilage of the joint was so marked in some places that the subchondral bony lamellae were denuded and at times even showed defects. It was interesting to observe that these defects sometimes again become repaired by a filling of a tissue which was at a stage between fibrous bone and calcified fibrous bone and calcified fibrous cartilage. So there were reparatory processes going on beside destructive changes. Altogether the pathologic picture was more quiescent. The proximal fragment of the big toe showed changes similar to those of the distal fragment as far as the bony structure was concerned. It was clearly shown that the present cortex derived its formation from a periosteal osteophyte of which the vertical arrangement of vessels was characteristic.

Whereas the distal end was more or less well preserved in its outline, the proximal end, which was supposed to articulate with the metatarsal bone, was destroyed to such a degree that there was nothing which would even suggest that once a joint was present. The bone ended rather abruptly with the diaphysis, with lacunar resorption at the outside of the cortex. The marrow cavity was open, and toward the proximal end the bone marrow showed more and more fibrosis, with hyperemia and some chronic lymphocytic infiltration, and blended gradually into an extensive area of scar tissue occupying the place where previously the metatarsophalangeal joint was present. There was almost nothing left of the metatarsal bone. An irregular piece of bone was included in the scarred soft tissue. The end of the joint could not be made out. The surface was still in an active phase of osteoclastic resorption. The bone was made up chiefly of lamellar bone tissue, quite sclerotic, and contained marrow spaces filled with fibrous bone marrow. The process appeared to be an attempt at regeneration of bone without gaining the real shape of a metatarsal bone.

Second Toe: The cutaneous changes did not reveal any new features. Here again the number of sweat glands was surprising. The callus on the plantar side corresponding to the metatarsophalangeal joint was so extreme that the specimen had to be trimmed for purposes of cutting. The hyperkeratosis was marked. On gross inspection all the constituents of the toe were present. The metatarsal bone and first phalanx were considerably misshapen, but the second and third phalanges were markedly osteoporotic. The interphalangeal joint between the second and the third phalanx showed the cartilage to be well preserved. The changes caused by disuse of the joint in the first interphalangeal joint were more marked. The first and second phalanges formed with their long axis an angle of about 90 degrees, with slight plantar subluxation of the second phalanx on the first. Owing to this displacement the dorsal capsular structures were kept tight and exerted some pressure on the convex surface of the joint at the distal end of the first phalanx. This pressure combined with a lack of play in the joint brought about a pronounced thinning out of the cartilage of the joint in this special area. At the margin of the joint almost the entire noncalcified cartilage had disappeared. A cellular layer of fibrous tissue had formed, which included some polymorphonuclear cells (chondroclasts), busy with the resorption of the cartilage of the joint. The pressure, however, also had influence on the bony portion of the epiphysis in that area, where the intra-articular part was covered only by the periosteum and the synovial tissue. Here was marked osteoclastic resorption on the articular surface, with apposition of bone at the endosteal surface. This process of combined resorption and apposition led to a gradual deformity of the epiphysis, with flattening of that portion of the convex end of the joint which was out of contact with its antagonist and under pressure from the side of the tightened capsule.

Compared with the pronounced osteoporosis of the distal portion of the toe, the proximal part of the first phalanx and the entire metatarsal bone were in striking contrast, as they showed marked osteosclerosis. This osteosclerosis represented a healing stage in an inflammatory osteomyelitic process involving the proximal end of the first phalanx and the metatarsal bone. At the advanced stage of the process it is of course difficult to determine where the inflammation took its origin. It seemed, however, that the origin was in the metatarsophalangeal joint, and from there the adjacent bony portions were gradually involved.

The metatarsophalangeal joint was completely destroyed in its normal outlines and tissues. All that was present was an extensive area of scar tissue which extended from between the phalanx and the head of the metatarsus down to the

plantar surface of the toe. It was clear that this extension of scar tissue was a result of the healing of a sinus tract running down to an ulcer of the toe. But the scar tissue was by no means uniform or homogeneous. On the contrary, between the metatarsus and the phalanx, an irregular cleft with side branches was noted. The walls of the cleft formed by dense fibrous tissue tended to the formation of villi and even formed some synovial endothelium. This irregular cleft could be interpreted as a joint space formed secondarily in scar tissue, which fitted the place of the metatarsophalangeal joint after its destruction. It was a form of nearthrosis illustrating nature's attempt to maintain a joint in the metatarsophalangeal region. That this metatarsophalangeal joint was destroyed by a process quite different from that which led to the changes in the interphalangeal joints as described previously can be seen from the following observations:

1. The ends of the joint of the first phalanx and the metatarsal bone were entirely involved in a pathologic process. No normal hyaline cartilage was left. The cartilage covered the bone tissue irregularly and even extended continuously into the marrow spaces. This alone showed that there must have been a pronounced destructive process which led to the destruction of the ends of the joint. There was haphazard and incomplete regeneration of bone tissue. At the site of the metatarsus the regeneration was hyperplastic and abundant, whereas at the proximal end of the first phalanx it was deficient, leading to a diminution of the proximal end. The bone tissue found on each side was represented by spongy bone without static structure. It formed within fibrous bone marrow which was still hyperemic and showed here and there infiltration by chronic inflammatory cells. The bony trabeculae were in the stage of apposition. Thick layers of osteoblasts and osteoid bone could be seen almost everywhere; osteoclastic resorption inside the bones was relatively scarce; it was marked, however, at the surface of the periosteum. Toward the scar tissue occupying the place of the metatarsophalangeal joint the bone tissue dissolved gradually the more isolated bony trabeculae. There were no continuous bony lamellae to separate the marrow spaces from the scar tissue.

2. The surrounding tissues, including scar tissue, frequently showed chronic inflammatory changes, with more diffuse or more circumscribed infiltration by lymphocytes and plasma cells.

3. There was one focus close to the metatarsal bone and, where included in scar tissue and surrounded by a form of fibrous tissue capsule, a small sequestrum could be seen lying in well preserved purulent exudate. This made us sure that this was the site of a formerly virulent suppurative process which at the time of observation appeared extinct. But to judge from this small focus of acute infection it would not have needed much stimulation for a flare-up.

As far as the bony structure of the metatarsal bone and the first phalanx was concerned, the typical picture of inflammatory osteosclerosis was present, as was well described by Scaglietti.

Third Toe: The cutaneous changes of the third toe were essentially the same as those of the other two toes. The first interphalangeal joint showed fibrous ankylosis, shrinkage of the capsule of the joint with obliteration of the peripheral portion of the cavity of the joint and thinning of the cartilage of the joint with subluxation by a pannus-like formation of fibrous tissue.

The metatarsophalangeal joint was gone, leaving no trace. Dense scar tissue occupied its former site, running down to the plantar surface of the skin, with hyperkeratosis. The scar tissue was rich in vessels and showed chronic cellular infiltration. In some areas it showed even older granulation tissue, with dense

infiltration by lymphocytes and plasma cells. A few muscle cells were included in the scar tissue, with well preserved transverse striation. These changes showed that a short time previously in this area a more active suppurative process had been present which had quieted down.

Proximal to this area was an extensive massive formation of cartilaginous and bony tissue, which resembled in many respects the picture of osteochondroma or myositic ossifications. In this instance it probably represented extensive callus formation or, better, an attempt at regeneration of the metatarsal bone, which had been almost entirely destroyed. There was hyaline cartilage of a more embryonal type, in which the cells toward the periphery were more and more swollen (spherical), and the ground substance became calcified. Then from the surrounding fibrous tissue osteoclasts absorbed calcified cartilage, and apposition of bone tissue took place at the surface of the calcified cartilage. Thus, spongy bone formed, which gradually became transformed into bone tissue of a more mature appearance. Calcification of cartilage and formation of bone tissue were so extensive that in the roentgenogram a dark shadow was cast.

The distal, small part of the metatarsal bone was present as a bony portion, but no normal bony structure was seen. Fibrous bone marrow was densely infiltrated by chronic inflammatory cells. The bony trabeculae were of irregular structure, showing many blue cement lines, which frequently ran parallel to each other and to the surface of the trabeculae. The structure of the bony trabeculae became more complex the more one approached the area of the bony and cartilaginous callus formation. The marrow spaces showed dense infiltration, and among the infiltrated cells polymorphonuclears were by no means rare. The bony trabeculae were connected with each other by newly formed fibrous bone tissue, which was laid down in such an amount that the old bony trabeculae were covered from all sides with new bone tissue and the marrow spaces were almost entirely occluded. The old bony trabeculae were alive. This picture again resembled that of metastasis of osteoplastic carcinoma, and in this instance it was due to sclerosing osteomyelitis. The osteosclerotic bone tissue surrounded an area interposed between the callus formation and the rest of the metatarsal bone. In this area many foci of puslike exudate were undergoing the process of organization. They became surrounded by loose fibrous tissue. In some places within the exudate small necrotic bony fragments were seen, sequestrums which either floated frequently in pus or became resorbed by polymorphonuclear leukocytes. Here again the picture was that of a subacute or even a chronic inflammatory lesion. Heretofore acute inflammatory signs had been minimal.

The proximal surface of the joint of the metatarsal bone was relatively well preserved. The hyaline cartilage of the joint was thinned out and became gradually resorbed from the margins, but there were no signs of necrosis and there was no more marked erosion of the cartilage.

Fourth Toe: The changes were essentially the same as those noted in the third toe, except that there was a disklike fibrous tab extending into the joint as an interposition from the shrunken capsule at the distal interphalangeal joint. The metatarsophalangeal joint was gone, and the area was supplanted by dense scar tissue. There was a fracture of the shaft of the metatarsal bone, with no signs of healing.

Fifth Toe: The most marked changes were seen in the metatarsal bone, in the joint and in the proximal end of the proximal phalanx. The shaft was almost entirely gone, and the degenerative changes predominated, with no signs of acute inflammation.

Brief reports of five other cases are included.

CASE 2 (fig. 3).—Edith S., 4 years of age, was admitted to the hospital in May 1922 with a diagnosis of spina bifida, congenital calcaneovalgus, perforating ulcer and osteomyelitis of the os calcis.

The child was under observation over a period of eight years. Her case illustrates the effects of pressure due to the deformity of the calcaneum on the basis of a myelodysplasia of the spinal cord, as evidenced by the complete cleft between the sacrum and the first lumbar vertebra. Ulceration followed blistering and

Fig. 3 (Edith S., case 2).—Roentgenograms showing the effects of pressure caused by deformity of the calcaneum, attributed to myelodysplasia of the spinal cord.

persisted intermittently over the entire period of observation. In 1924 the os calcis was operated on by the split heel approach according to the Orr method. The wound healed, although necrotic bone was encountered at operation. Sensation was always intact. Ulceration recurred later.

Examination of one section revealed chronic osteomyelitis with inflammatory osteosclerosis, with a tendency toward healing. The section of skin was remarkable for the formation of an epithelial cyst undoubtedly of traumatic origin. Owing to the ulceration of the skin, displacement of epithelium into the deeper portion of the subcutaneous tissue accounted for the formation of a cyst.

CASE 3 (fig. 4).—Albert P., aged 53, was admitted to the hospital in October 1933. He had bruised his foot on a cinder six months previously, while wearing thin-soled shoes. An open sore soon developed, which healed and reopened. At the time of admission to the hospital there was drainage of three months' duration. Three years previously the patient had experienced lancinating pains in the legs.

Examination revealed an ulcer on the ball of the foot, with foul discharge. The entire foot was swollen, discolored, cold and clammy. The roentgenograms strongly suggested the changes that occur in cases of leprosy. The metatarsophalangeal joints and their contiguous bony elements were typically destroyed and were undergoing resorption. The reaction to the Wassermann test was negative. The sugar content of the blood was 297 mg. per hundred cubic centimeters. A diabetic regimen was indicated. Permission for amputation was refused.

Fig. 4.—The upper roentgenograms show compound fracture of the first metatarsal bone, necessitating resection. This type of direct infection does not produce the general changes noted in cases of perforating ulcer. Compare them with the lower roentgenogram (Albert P.), in which the changes suggest those that occur in leprosy. The metatarsophalangeal joints and their contiguous bony elements are typically destroyed and are undergoing resorption.

Case 4 (fig. 6).—Fred N., aged 33, was admitted to the hospital in August 1933. The patient had always had difficulty with his feet but did not have real pain at any time. Six months previously an ulcer appeared at the end of the second toe on the right foot, which healed after a piece of dead bone was removed. The third toe was similarly affected one month after this, and at the same time a blister developed on the second toe of the left foot. The patient opened the blister, and an ulcer developed, which persisted. There was no history of trauma or freezing. The patient walked fairly well.

Examination revealed an ulcer the size of a dime on the ball of the big toe of the left foot which was sharply demarcated but rather ragged. Part of the nail was missing on the big toe of the right foot, and the entire toe was short. The third toe of the right foot was thicker than that of the left foot and had a

Fig. 5.—Recurrent perforating ulcers of the toes and heels in a young woman subsequent to an attack of epidemic encephalitis and associated with osteomyelitis of the phalanges. These episodes were all due to combined nervous and peripheral vascular disturbances which were uninfluenced by lumbar ramisection performed bilaterally about eight years before the photograph was taken. In the photograph the ulcers have healed, as they always did following a period of rest and relief from weight bearing.

small ulcer at the distal end. The ulcers were painless. The neurologic examination gave negative results.

Roentgenograms showed spina bifida occulta of the first sacral segment. The anteroposterior views of the feet revealed marked hallux valgus of the right foot, with some subluxation of the proximal phalanx. There was a defect of the third phalanx of the second toe, with no intrinsic pathologic process of the right foot.

The first phalanx showed increased width. There were some irregular periosteal thickening and proliferation, with the formation of small exostoses. There was no definite swelling of the soft tissue of the big toe of the left foot.

Dermathermic readings after injection into the posterior tibial nerve showed the basic temperature on the right side to be 29.6 C. and after thirty minutes 30.9 C., and on the left side, 29.9 C., and after thirty minutes 29 C.

Treatment started on September 1 included resection of the ulcer of the big toe and plastic closure, with some improvement. On October 25 a Mayo bunionectomy was performed on the left foot. The wound healed. A pathologic examination of soft tissue removed at operation revealed a torpid cutaneous ulcer with hyperplasia of the sweat glands and slow and irregular epithelization from the margins. The diagnosis was early perforating ulcer of the foot caused by circulatory insufficiency, with few changes in the bone.

Case 5 (fig. 7).—Isabell M., aged 6 years, was admitted to the hospital in March 1927. The child was born with a meningocele over the lumbar region and a club foot (left). The meningocele was surgically removed. The roentgenogram showed a complete lumbar and sacral cleft. In 1927 a small ulcer

Fig. 6 (Fred. N.; case 4).—The anteroposterior views of the feet revealed marked hallux valgus of the right foot, with some subluxation of the proximal phalanx. There was a defect of the third phalanx of the second toe, with no intrinsic pathologic process of the right foot. There was no evidence of abnormality of the spine.

developed on the lateral surface of the ball of the foot, which was painless and healed slowly. In 1929 the foot was more markedly deformed, and the ulcer again appeared and persisted. This was on the lateral weight-bearing surface. Surgical correction and stabilization of the foot was performed. The operative wound healed, but the ulcer had not after some months. Two years later all signs of ulceration had disappeared, and the fusion of the foot held firmly. In 1932 an ulcer developed on the big toe of the same foot, with some moderate drainage. This continued until osteomyelitis set in in the proximal phalanx. Operation was performed according to the Orr method. The bone was friable, fragmented and sequestrated, and there were considerable reddish granulations but no free pus. The wound healed in about eight months. Staphylococcus was recovered from the wound at the time of operation.

Histologically the bone showed lacunar absorption, and the tissue between the trabeculae was filled with granulation tissue densely infiltrated by inflammatory

cells, of which the polymorphonuclears predominated. The diagnosis was chronic osteomyelitis, with a perforating ulcer of the foot.

Case 6 (fig. 8).—Earl T., aged 33, was admitted to the hospital in March 1933. Ulceration followed local injury to the foot three years previously. There were some pain, heat and redness over the bunion area for several days before drainage set in at the outset of the formation of the ulcer. The area healed with rest after two weeks. In October 1932 there was a recurrence, with a plantar lesion over the same area. During the same winter the opposite foot became similarly involved. Some swelling of the ankles persisted with prolonged weight bearing, because the patient did not stop working. Both feet had been frozen five years previously.

Fig. 7 (Isabell M.; case 5).—The roentgenogram of the spine shows a complete lumbar and sacral cleft. The other roentgenogram is of the club foot (left).

The family history revealed that the mother suffered from callosities. A maternal aunt was similarly affected, and her daughter had a painful condition of the foot which recently required the removal of a small piece of bone.

Examination revealed the right foot and leg to be edematous, and the fore part of the foot was especially red on the dorsum. There was pitting edema, but the foot was not tender. Under the large toe was an ulcer ¾ inch (1.9 cm.), with considerable oozing. Two smaller toes had dark shiny calluses which looked like areas of beginning dry gangrene. There was a marked hallux valgus on the left foot; the nails were hypertrophied, and the sole had two calluses, in one of which was an ulcer. The reflexes were slightly hyperactive.

The laboratory examination gave negative results.

The dermathermic reading after thirty minutes showed an increase of 0.8 C. Before injection the temperature was 28.7 C. The anteroposterior roentgenogram of the feet showed an enormous widening of the anterior portion of the arches. The first metatarsal bone of each foot was in a position of marked adduction. There were destructive lesions at the distal end of the fifth metatarsal bone of the left foot, on the distal two thirds of the first phalanx of the big toe of the right foot and on the head of the metatarsal bone of the second toe of the right

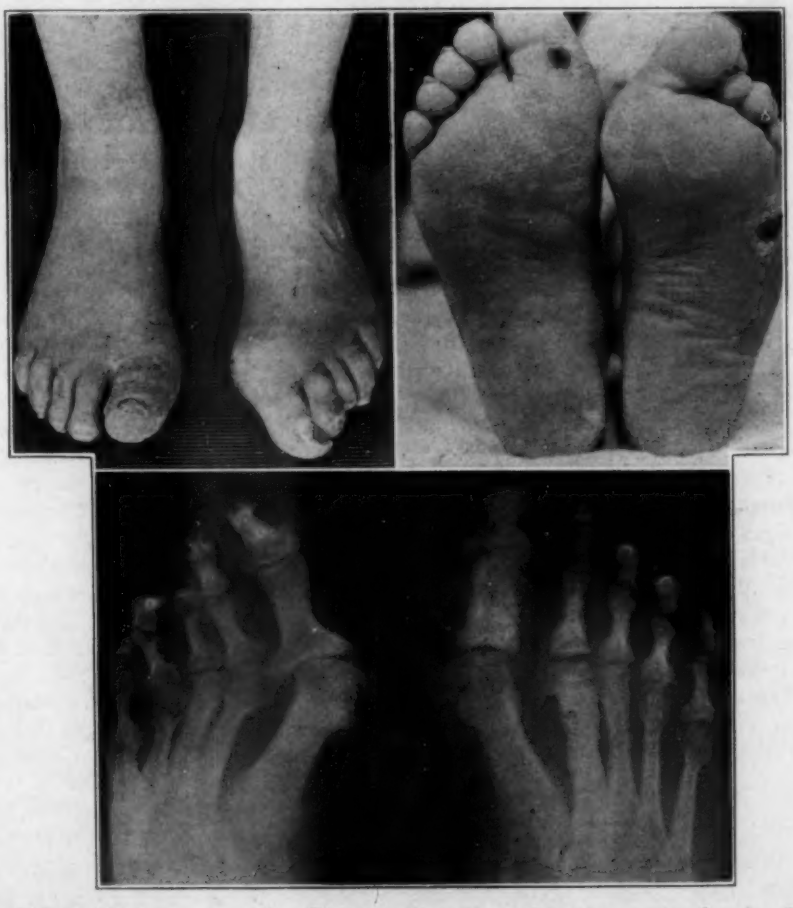


Fig. 8 (Earl T.; case 6).—Photographs and roentgenogram showing involvement of both feet following local trauma to one foot three years previously.

foot. There were destructive lesions of the second interphalangeal joint of the big toe of the right foot and of the fifth toe and of the metatarsophalangeal joint of the fifth toe of the left foot. There were peculiar destructive processes at the distal end of the end phalanx of each toe of the left foot. Periosteal reaction was present along the diaphysis of the third and fifth metatarsal bones of the left foot, and of the first, second and third metatarsal bones and along the first phalanges of the first and the second toe of the right foot. There was marked swelling of

the soft tissue of the big toe of the right foot; the roentgenogram showed activity in the first and second phalanges, whereas all the other lesions seemed to be of longer duration and in a more chronic stage. The distal end of the fifth metatarsal bone of the left foot was completely destroyed, and there was subluxation of the toe in a dorsal direction. The lesion was that of an osteomyelitic process of the metatarsal bone and of the phalanges, including the joints. A specimen was removed for biopsy, consisting of the proximal phalanx of the big toe and the heads of the metacarpal bones. There were edema and thickening of the tissues. The periosteum was thickened and adherent. Concentric lamellations were noted on removing the cortex. The bone was hemorrhagic, and a definite medullary cavity was not noted. The wounds healed in four days by first intention. On May 18 treatment was instituted with a whirpool bath and irradiation with ultraviolet rays, with considerable improvement. Examination on October 6 revealed the ulcers to be healed, but in July that on the big toe of the right foot reopened, and one week later that on the left foot reappeared. These were present with some swelling at the time of writing.

A piece of skin and three small pieces of bone taken from the first metatarsal bone were examined histologically. The piece of skin showed marked thickening of the subcutaneous layer, which was entirely composed of fibrous tissue. The subcutaneous fat tissue had completely disappeared. Chronic cellular inflammation was present in some places but did not form an outstanding feature. The pieces of bone taken from the first metatarsal bone showed irregular but dense spongy bone. The bony trabeculae were composed of fibrous and lamellar bone of almost equal amount. They were included in dense and hyperemic fibrous marrow. The bone tissue seemed in a quiescent stage. There was not much resorption or apposition of bone. The new bone was almost entirely the result of formation of fibrous bone originating in the fibrous bone marrow. There were no signs of acute inflammation.

Several small pieces of bone taken from the second metatarsal bone showed essentially the same picture as that of the first metatarsal bone. Fibrous bone marrow was present, with many hyperemic vessels. The spongy bone was even more irregular than that in the first metatarsal bone; especially was lacunar resorption in the marginal portions of the metatarsal bone very mixed.

The diagnosis was chronic osteomyelitis of the first and second metatarsal bones without signs of a specific pathologic process.

SUMMARY

We have discussed the general features and the changes in the bones in cases of malum perforans pedis as based on observations on a series of 33 cases. It is evident that any treatment to be effective must be aggressive and must be instituted almost early enough to be considered as a prophylactic measure. Therapeutic methods must aim at rectifying any or all underlying constitutional causes that are operative, as well as the local condition. It is safe to assume that any improvement in the circulatory status of the affected member will react favorably on such skeletal changes as are more or less collateral and which consist chiefly of degenerative changes due to atrophy of disuse and local pressure at the joints. There is a marked tendency to repair and regeneration. It is suggested that the pavaex treatment offers an excellent means of

obtaining an improvement in the local circulation. Those areas which are definitely osteomyelitic must be considered as involved in primary direct inflammatory changes superimposed on the basic degenerative transformations and are due to infection directly by way of the break in the surface of the skin. This is supported by the fact that in a series of over 130 cases of osteomyelitis of the small bones of the foot the condition in the vast majority was due to a perforating injury. In the series of over 1,500 cases of osteomyelitis there were only 8 instances of peripheral metastases to the bones of the foot. Therefore we believe that all osteomyelitic foci in perforating ulcer of the foot should be radically attacked as in any other osteomyelitic localization. There must be a careful clinical evaluation of the bony changes as seen in roentgenograms in order to distinguish the osteomyelitic from the collateral changes. The presence of sequestrums is of great aid in forming an accurate opinion and indication. Amputation should be resorted to only after all else has failed and with the thought that ulceration has been known to recur in the stump and also in the other foot after it has taken up its added static burden.

RÔLE OF THE ILEOCECAL SPHINCTER IN CASES OF OBSTRUCTION OF THE LARGE BOWEL

LOUIS SPERLING, M.D.
MINNEAPOLIS

CONTENTS

Review of literature
- Anatomic structure of the ileocecal sphincter
- Nerve supply
- Competency of the sphincter

Experimental work
- Study of competency of the ileocecal sphincter in cadavers
- Tests of competency of the ileocecal sphincter in dogs
- Tests of competency of the ileocecal sphincter in man by insufflation of air under low pressure and by the administration of barium enemas

Significance of the competence of the ileocecal sphincter in colonic obstructions
- Reports of cases
- Effect of increased intra-enteric pressure on the wall of the bowel
- Experimental work

Measurement of the intra-enteric pressure in cases of obstruction of the large bowel
Measurements of intra-enteric pressure in clinical cases of obstruction of the large bowel (table)
Comment
Conclusions
Bibliography

It has been known for a long time that distention of the small bowel is rarely observed in cases of obstruction of the colon. The older German writers, Kreuter, Anschütz and others, have explained this observation by assuming that the ileocecal sphincter is competent to withstand back pressure. The literature from 1890-1900 is replete with reports of cases of obstruction of the colon in which gangrene and perforation eventually developed with absence of distention of the small bowel. The more recent literature on obstruction (Burget and Gatch and their associates and others) has stressed the fact that changes in the wall of the bowel are more likely to occur in cases of obstruction with a closed loop than in those of simple type.

The importance of the ileocecal sphincter in obstruction of the large bowel depends entirely on whether it is competent to prevent backflow into the ileum, thus converting an otherwise simple type of obstruction into a closed loop.

From the Department of Surgery of the University of Minnesota.

Submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science (September 1934).

This thesis deals with the question of why obstruction of the large bowel, especially that due to carcinoma of the left half of the colon, is of a closed loop type and whether the ileocecal sphincter is sufficiently competent to prevent regurgitation of gas and fluid into the small bowel until the increase of intra-enteric pressure is great enough to compromise the circulation of the wall of the large intestine.

The work is divided into four parts:

1. Experimental study of the competency of the ileocecal sphincter in cadavers, dogs and clinical subjects.

2. Observations on the intra-enteric pressure which may obtain in clinical cases of obstruction of the large bowel.

3. Experimental study to determine the effect of such recorded pressures on the structure of the wall of the bowel.

4. Presentation of clinical cases.

The value of this work is twofold. First, it emphasizes that obstruction of the large bowel should be treated as a closed loop or a potential strangulating type. Second, for the first time measurements of the increased pressure which obtains in cases of obstruction of the large bowel are recorded.

REVIEW OF LITERATURE

An enormous literature has accumulated concerning the pathologic physiology of intestinal obstruction, but it has dealt mostly with the variety seen in the small bowel. Relatively little attention has been paid to obstruction of the colon, and a tendency to regard it in the light of recent observations on ileus of the small bowel is evident. The difference between ileus of the small bowel and that of the large is chiefly one between simple obstruction and that of the closed loop type, and it depends on the presence of a competent ileocecal sphincter, which prevents backflow and allows intra-intestinal pressure to mount to dangerous heights.

Bauhin described the ileocecal sphincter in 1605 and conjectured that it was competent to resist back pressure, acting chiefly in the mechanical rôle. This was accepted for about three hundred years without question. However, in the last fifty years much has been said and written with reference to the competency of the ileocecal sphincter to withstand the injection of fluids or the insufflation of gases into the rectum.

That the valve acts not only mechanically but as a true sphincter was emphasized by Cannon and also by Rutherford, who demonstrated the presence of circular fibers. Balli through important histologic studies not only confirmed the work of Rutherford but demonstrated the presence of oblique fibers. The valve acts then as a real sphincter, similar

to that of the pylorus, and regulates the passage of chyme from the small bowel into the large and effectually prevents regurgitation.

Elliot has shown that the ileocecal sphincter is not an essential organ; i. e., it is absent in the bear, ferret and hedgehog. It is not essential to life, as patients on whom resection of the ileocecal region and ileocolostomy have been performed apparently accommodate themselves to the change after a short period of diarrhea and experience no further trouble.

ANATOMIC STRUCTURE OF THE ILEOCECAL SPHINCTER

In textbooks of anatomy (Sobotta and McMurrich) the ileocecal sphincter is described as formed by two folds of mucous membrane, which are termed the upper and lower lips of the valve. The deeper layers of the lips are the two muscular layers of the ileum. The fold appears as a hemispherical papilla 1.8 cm. in diameter, projecting 1 cm. above the cecal wall. The surfaces of the lips directed toward the large intestine have the character of the mucous membrane of that portion of the intestine, while those that are turned toward the lumen of the ileum are covered with villi to the edges of the valvular orifice. When open the lips of the valve are separated by an elongated slit pointed at either end, and when in contact they form a complete closure between the large and the small intestine. From the lips of the valve semicircular folds radiate to the anterior and posterior portions of the wall of the cecum. They are termed the frenula (anterior and posterior) of the valve, and they form the boundary between the cecum and the ascending colon.

NERVE SUPPLY

Elliot has demonstrated that the motor innervation of the sphincter is through the ninth to the twelfth thoracic nerve and the first and second lumbar segments of the splanchnic nerves. He obtained contraction by stimulation of these nerves and by painting the sphincter with epinephrine. Elliot's observations were confirmed by Hromada and by Dale. Stimulation of the vagi and the pelvic nerves did not result in contraction. Walcker cut the ileocolic nerves, and paralysis of the sphincter resulted. The sphincter becomes incompetent after section of the spinal cord (Elliot). Tönnis cut the postganglionic fibers to the ileocolic and midcolic arteries and the rami communicantes of the twelfth and thirteenth nerves on each side and noted paralysis of the sphincter. There was also paralysis if the splanchnic nerves were cut in the chest. Hinrichsen and Ivy studied the innervation of the ileocecal sphincter and came to the following conclusions:

1. The sphincter acts physiologically.

2. Stimulation of the vagus produced first relaxation, followed by a strong contraction; therefore, the vagus has both motor and inhibitory fibers.

3. Stimulation of the splanchnic nerves resulted in contraction only; i. e., the splanchnic nerves maintain the tone of the sphincter.

4. Contraction may occur following stimulation of almost any abdominal nerve and from distention of the stomach, duodenum or colon.

5. Contraction during the feeding reflex is due to a duodenocolic reflex.

The effect of the duodenocolic or the gastrocolic reflex on the sphincter has been mentioned by several writers. Hannes found that the ileocecal sphincter is open in dogs except after the intake of food. Tönnis showed that when the stomach is full the sphincter remains closed and that it opens only with the onset of peristalsis in the ileum. Examination one hour after a meal seldom showed the presence of incompetency. From this it would appear that filling of the ileum by a barium enema is no index of insufficiency if the enema is taken on an empty stomach during fasting or long after a meal. That the nerve control of the sphincter is chiefly through the local portion of the intramural plexus of Auerbach was shown by Short, who observed in experimentation on a man with the ileocecal sphincter exposed through a cecostomy opening that stimulation of the folds of the cecal mucous membrane when it was in full activity after a meal delayed outflow from the ileum and made the jets less frequent, though it did not stop them altogether. It is conceivable that the irritation set up by distention of the cecum and stasis of fluids and gases due to stricture of the left portion of the colon would result in increased tone of the sphincter, making it more competent in a person with obstruction than in a normal person.

From the experiments of Heile it seems that filling of the colon causes a firmer contraction of the sphincter, for in experiments performed for a different purpose he showed that the introduction of a tampon cannula into the colon produced a much slower evacuation of the small bowel through the appendical fistula. This may be due to increased contraction of the muscle fibers of the sphincter after mechanical irritation produced by gradual filling of the colon. Sudden massive filling of the colon, such as occurs with the barium enema, is not to be compared with the gradual distention in cases of obstruction of the large bowel.

Case stated that reversed waves are the prevailing type of movement in the proximal portion of the colon and that they may become exaggerated in the presence of obstruction of the left half of the bowel resulting in stasis in the proximal segment. He called attention to the fact that many writers have described instances of gangrene of the cecum associated with obstruction of the pelvic portion of the colon, and he explained the phenomenon as due to waves of antiperistalsis originating at a ring of tonic contraction, which create back pressure and result in

distention of the cecum. Blamoutier definitely observed this ring of contraction from which waves of antiperistalsis seemed to originate. He commented on the fact that the ileocecal sphincter would soon atrophy if it were not for the presence of these reversed waves.

COMPETENCY OF THE SPHINCTER

In 1586 Piccolhomini showed that injection of air or water through the rectum produced closure of the ileocecal sphincter. Fabricius ab Aquapendente (1618) was unsuccessful in injecting air through the rectum into the ileum. Postmortem tests of incompetency of the sphincter have been made by many investigators. Rutherford summed up the results of these tests as follows: "Want of uniformity in description makes it probable that the ileo-cecal valve changes soon after death." He concluded that normally the valve is competent during life. The fact that the ileocecal valve acts as a muscular sphincter makes it evident that its competency must depend on the tonicity of its muscle fibers and that all factors influencing the tone of the ileocecal region, whether of local or of remote origin, also regulate the sphincter.

On the assumption that the valves act mechanically only, Plesch compared them to a pair of lips and indicated that there would be no incompetency when the lips were accurately apposed to each other.

The degree of increased colonic pressure required to force the ileocecal sphincter has been investigated by many workers. Cannon noted that in a cat large enemas were passed into the small bowel but that small enemas were not. Von Genersich forced from 6 to 9 liters of water into the colon under a pressure of from 80 to 100 cm. of water and found that the fluid passed into the small bowel. He observed this in both living and dead subjects. This was confirmed by Dauriac. Von Ziemssen forced carbon dioxide into the colon in both man and the dog. In dogs he was able to recover it from the mouth; in human subjects he recovered it only when a high pressure was used. Senn found that air under a pressure of 2.5 pounds (1.1 Kg.) per square inch (6.45 sq. cm.) could be passed from the anus to the mouth in man. However, the ileocecal sphincter withstood a pressure of 0.7 Kg. successfully, and only when the pressures exceeded 0.9 Kg. was colo-ileal regurgitation evident. In his experiments on dogs he concluded that the ileocecal sphincter resisted a pressure of from ½ to ¾ pound (0.2 to 0.3 Kg.). In experiments in which fluid was forced through the ileocecal valve, multiple lacerations of the peritoneal surface of the large bowel were shown at autopsy. Walcker inflated the cecum in cadavers and found that the sphincter withstood a pressure of from 2 to 80 mm. of mercury. In 1 specimen the valve withstood the increase of pressure until the cecum ruptured. Burt determined by the introduction of air

the pressure required at various levels to rupture the bowel. He found that the cecum had the least resistance, rupturing at a pressure of 1.53 pounds (0.69 Kg.) per square inch (6.45 sq. cm.). Joltrain, Baufle and Coope (cited by Larson and Bargen) produced pressures of various degrees in the large bowel and noted the effects. They found that the normal colon could hold about 1,000 cc. of fluid, which they computed was under a pressure of from 16 to 20 cm. of water. When the pressure was increased to about 30 cm. of water with a volume of 3,000 cc. in the colon the patient complained of pain and distress.

Heile stated that a pressure of 20 mm. of mercury is the highest that will be withstood by the ileocecal sphincter, although in some cases he observed rupture into the peritoneal cavity.

The percentage of cases of insufficiency of the ileocecal sphincter as reported by various roentgenologists varies from 0 to 90 per cent. Groedel has never seen incompetence of the valve in withstanding contrast mediums in healthy persons. Drummond and Schwartz noted that enemas rarely pass beyond the cecum. In a series of 1,500 cases in which a barium enema was given Case reported 250 instances of insufficiency. Other values are those cited by Hammer (70 per cent), Herlyn (42 per cent), Alvarez (60 per cent), Carman (90 per cent), Dietlen (20 per cent) and Cole (33 per cent). This variance in values must indicate a variance in technic in the administration of the barium enemas. It is reasonable to assume that the higher the pressure utilized the greater the percentage of instances of incompetency. At the University Hospital it has been estimated by Dr. Rigler that in approximately 80 to 90 per cent of all the cases in which barium enemas are given, incompetency of the sphincter is shown.

Most investigators have found that the sphincter is competent to withstand moderate increases of intra-enteric pressure. Rost stated: "In general, the valve prevents backflow from the large intestine and also remains closed when the pressure there is fairly high, as for example, in large intestinal ileus." Despite reports by roentgenologists on the relative insufficiency of the sphincter, the fact remains that in clinical cases of obstruction of the large bowel the condition is often observed to progress to gangrene of the cecum and perforation without evidence of dilatation of the small bowel. In an attempt to correlate these paradoxical statements the following observations are presented:

1. Pressures utilized in barium enemas produce sudden massive filling of the colon and are much higher than any which might occur even in cases of extreme obstruction. Filling of the colon in a case of obstruction is gradual. Forty millimeters of mercury (52 cm. of water) is the highest pressure I have measured in clinical cases of obstruction.

2. The pressure exerted by the barium enema is usually greatly augmented by manual manipulation of the cecum.

3. Barium enemas are usually given to persons while fasting, in which state, according to Tönnis, the sphincter is most likely to be incompetent.

4. The greatest percentage of barium enemas are administered to younger persons with a normal colon. The results cannot be applied to the pathologic colon subjected to gradual distention and increase of intra-enteric pressure, which must certainly, by irritation of the cecal mucous membrane, affect the tone of the ileocecal sphincter.

EXPERIMENTAL WORK

Experimental work was undertaken to ascertain: (1) whether the ileocecal sphincter is competent to withstand pressures which occur clinically in the colon; (2) whether irritation of the distal portion of the colon or of the ileocecal nerves will increase the tone of the ileocecal sphincter and make it competent to withstand higher pressures, and (3) whether increase of intra-enteric pressure to the limits observed clinically will result eventually in damage to the wall of the bowel sufficient to account for the clinical complications of gangrene, perforation and peritonitis. Clinical observations are reported of pressures that obtain in cases of obstruction of the large bowel.

STUDY OF COMPETENCY OF THE ILEOCECAL SPHINCTER IN CADAVERS

In the cadaver the ileocecal sphincter acts only mechanically, owing to the loss of tone. I shall refer to it as a "valve" in this section only. Throughout the remainder of the paper it will be referred to as a "sphincter."

The ileocecal portion of the bowel was obtained from 4 cadavers at autopsy. One specimen, obtained six hours after death, proved incompetent to withstand air injected through the ileum or colon. The second specimen, taken from a person who had died two hours before, was entirely competent (air could not be forced backward into the ileum under pressures of more than 144 mm. of mercury). Under this pressure, owing to distention of the colon, the air began to seep through the wall. Injection of air through the ileum under a pressure of 64 mm. overcame the resistance of the valve. Repeated attempts to inject air into the colon showed that the valve was competent to resist a pressure of 140 mm. In the third specimen, obtained two hours after death, a colo-ileal regurgitation was obtained at a pressure of 32 mm., and ileocolic insufficiency, at a pressure of 14 mm. In the fourth specimen, four hours after death, colo-ileal regurgitation was obtained at a pressure of 20 mm., and ileocolic insufficiency, at a pressure of 10 mm.

It would appear from these few studies on cadavers and from the literature already cited that observations on a cadaver are of no value, because, first, tone is an important factor in the competency of the

valve, as it acts like a true sphincter, and, second, competency of the valve in a cadaver would depend entirely on the degree of coaptation of the lips of the valve at the time of death.

TESTS OF COMPETENCY OF THE ILEOCECAL SPHINCTER IN DOGS

Dog 1.—With ether anesthesia, 1 foot (30.48 cm.) of intestine was isolated on either side of the ileocecal juncture and clamped with rubber-covered intestinal clamps. A T-tube cannula was inserted into the ileum and into the cecum 12 inches (30.48 cm.) distal to the sphincter, and one end was connected to a mercury manometer.

1. One hundred cubic centimeters of air was slowly injected into the ileum. The ileum was distended until a pressure of 70 mm. of mercury was reached. At this pressure the ileocecal sphincter suddenly relaxed and allowed air to enter the cecum.

2. The preceding experiment was repeated, the cecum being gradually filled with air until a pressure of 76 mm. was reached, when the sphincter became incompetent and allowed air to rush into the ileum.

3. Air injected into the ileum passed the sphincter only when a pressure of 72 mm. of mercury was reached. The colon became distended. When the ileum was suddenly decompressed the colon remained distended at a pressure of 15 mm., with the sphincter still competent. It was necessary to increase the pressure in the colon to 80 mm. again before the sphincter gave way and allowed air to pass into the ileum.

Dog 2.—Similar experiments were repeated at intervals of thirty minutes. The following results were obtained:

1. Ileocolic insufficiency appeared at a pressure of 54 mm. and colo-ileal regurgitation, at a pressure of 42 mm.

2. Ileocolic insufficiency was obtained at a pressure of 30 mm., and colo-ileal regurgitation, at a pressure of 32 mm.

3. Ileocolic insufficiency was obtained at a pressure of 40 mm., and colo-ileal regurgitation, at a pressure of 32 mm.

When the ileum was suddenly decompressed the sphincter held air in the colon under a pressure of 20 mm. of mercury.

Dog 3.—A T-tube cannula was inserted into the ileum, and a rectal tube was inserted into the colon through the anus and secured by a purse string suture. Air was injected through the rectal tube until a pressure of 226 mm. of mercury was recorded in the colon. At this pressure the sphincter evidently gave way, and air rushed into the ileum. Repetition of this experiment in thirty minutes gave the same result at a pressure of 60 mm. After this, complete paralysis of the sphincter ensued, the two systems acting as one.

In the rest of the experiments the effect of electrical stimulation with the induction coil of the splanchnic nerves (ileocolic vessels and the ileocolic nerve), the distal portion of the colon, the small bowel and other viscera was noted.

Dog 4.—Colo-ileal regurgitation occurred at a pressure of 36 mm. of mercury. When the ileo-colic vessels were stimulated the resistance of the sphincter was increased until it equaled a pressure of 124 mm. This test was repeated, and still

the resistance of the sphincter was equal to a pressure of 64 mm., almost twice that of the normal sphincter.

Dog 5.—Colo-ileal regurgitation occurred at a pressure of 40 mm. of mercury. When the distal portion of the colon was stimulated the resistance of the sphincter was equal to a pressure of 120 mm. In thirty minutes a redetermination of the normal resistance to colo-ileal regurgitation showed it to be equal to a pressure of 64 mm. On stimulation of the ileocolic vessels the resistance increased until it equaled a pressure of 107 mm.

Dog 6.—Colo-ileal regurgitation occurred at a pressure of 88 mm. When the distal part of the colon was stimulated the resistance of the sphincter was increased to withstand a pressure of 112 mm. The normal resistance was determined again and was found to be equal to a pressure of 66 mm. On stimulation of the ileocolic vessels a reading of 124 mm. was obtained before the sphincter gave way.

Dog 7.—Colo-ileal regurgitation occurred at a pressure of 60 mm. When the ileocolic vessels were stimulated the resistance to regurgitation was equal to a pressure of 114 mm. The normal resistance determined after thirty minutes was equal to a pressure of 50 mm. On stimulation of the distal portion of the colon resistance to regurgitation increased to withstand a pressure of 120 mm.

Dog 8.—The normal resistance to regurgitation was equal to a pressure of 70 mm. The resistance when the stomach was stimulated was still equal to a pressure of 70 mm. The normal resistance was again determined and found to be equal to a pressure of 76 mm. The resistance when the parietal peritoneum was stimulated was equal to a pressure of 80 mm. The normal resistance was equal to a pressure of 70 mm. On stimulation of the colon the resistance was equal to a pressure of 200 mm. Practically no change in the resistance to regurgitation was observed, except when the colon was stimulated.

A series of experiments on a cat was performed. Similar results were obtained as follows: The resistance to colo-ileal regurgitation under normal conditions was equal to a pressure of 70 mm. of mercury; on stimulation of the peritoneum, to 65 mm.; of the colon, to 78 mm.; of the ileocolic vessels, to 140 mm.; of the appendix, to 100 mm.; of the small bowel, to 50 mm., and of the colon, to 140 mm. The normal resistance again determined was equal to a pressure of 60 mm.

It is evident from these experiments that the ileocecal sphincter of the dog and cat is competent to withstand pressures of from 60 to 80 mm. of mercury. This normal resistance has a constant value for each animal, as shown by many repeated tests, and it is greatly increased by stimulation of the sympathetic nerves and by irritation of the serosa of the distal part of the colon. Stimulation of the parietal peritoneum of other viscera had no such effect. Stimulation of the colon must enhance the tone of the ileocolic sphincter through Auerbach's plexus. A similar observation was made by Short, who noted a decrease of ileocolic inflow when he stimulated the cecal mucosa. It is significant that even the normal resistance of the sphincter to colo-ileal regurgitation is much higher than any increased pressure noted in clinical cases of obstruction of the large bowel.

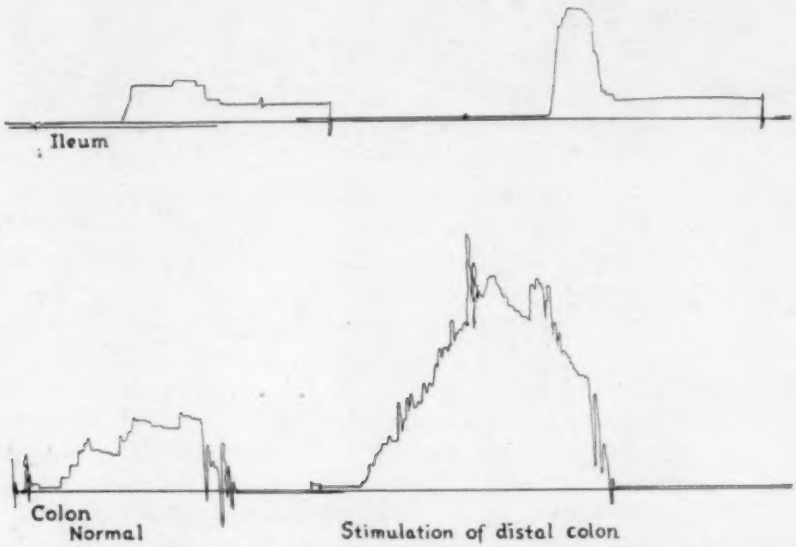


Fig. 1 (dog 5).—Increased competency of the ileocecal valve on stimulation of the distal portion of the colon.

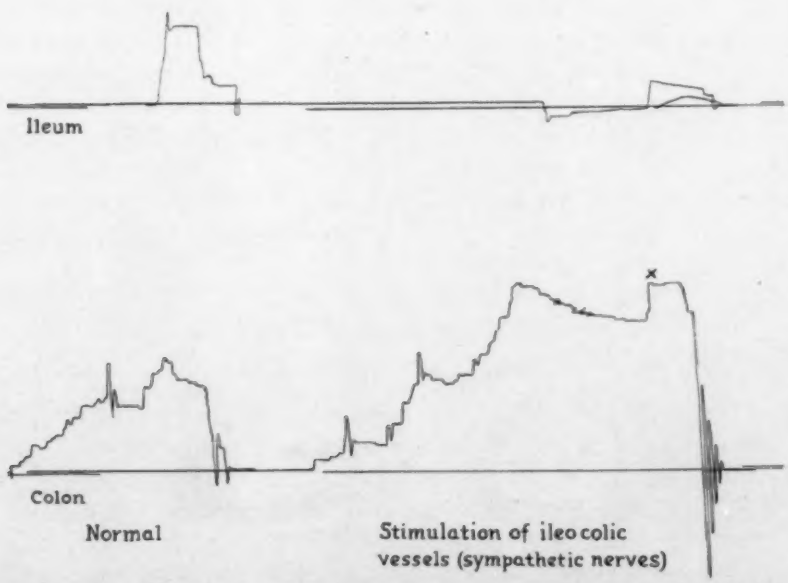


Fig. 2 (dog 7).—Increased competency of the ileocecal valve on stimulation of the ileocolic vessels (sympathetic nerves).

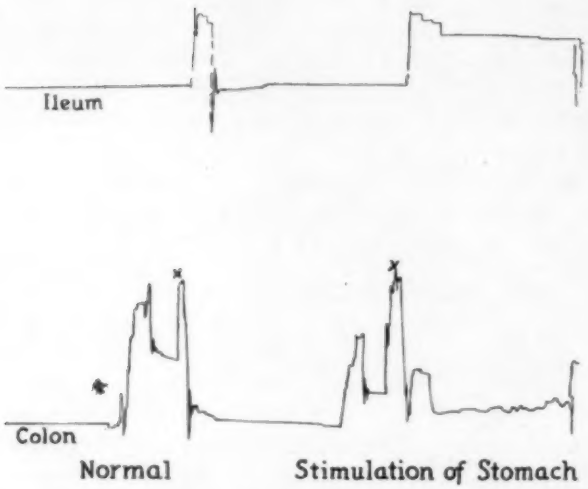


Fig. 3 (dog 8).—No increase in the competency of the ileocecal valve on stimulation of the stomach.

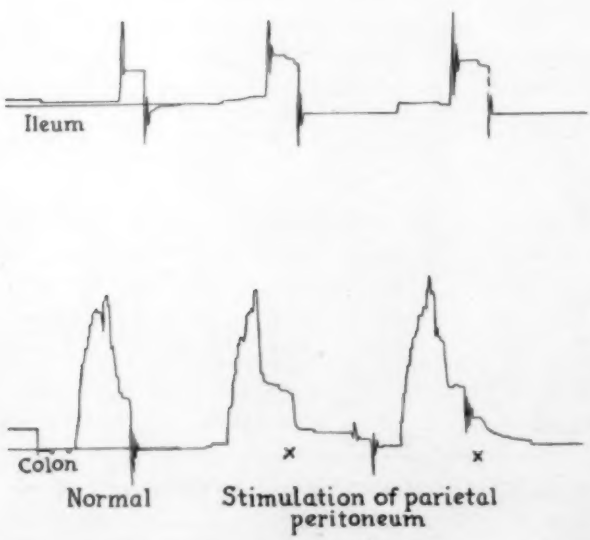


Fig. 4 (dog 8).—No increase in the competency of the ileocecal valve on stimulation of the parietal peritoneum.

TESTS OF COMPETENCY OF THE ILEOCECAL SPHINCTER IN MAN BY INSUFFLATION OF AIR UNDER LOW PRESSURE AND BY THE ADMINISTRATION OF BARIUM ENEMAS

With the cooperation of Drs. Rigler and Borman of the department of roentgenology of the University Hospital, it was observed that with the usual technic, i. e., with a pressure of gravity of 3 feet (91.44 cm.) of barium (from 80 to 100 mm. of mercury) and with manipulation of the abdominal wall, it was possible to render the ileocecal sphincter incompetent in a high percentage of persons. The competency of the human sphincter was tested under a lower pressure, 50 mm. of mercury or 2 feet (60.96 cm.) of barium. In a series of subjects the colon was first gradually distended with air until the cecum was well visualized under the fluoroscope. The barium was injected under a pressure of gravity of 50 mm. It was observed that the colon was much more difficult to fill with barium if air had previously been injected. This was interpreted as due to the competency of the ileocecal sphincter to withstand the compression of the air present in the cecum. In 5 cases the ileocecal sphincter was competent even after manipulation. In 3 instances the barium passed easily into the small bowel. In 1 case barium entered the ileum under a pressure of only 1 foot (30.48 cm.) of barium (30 mm. of mercury). In 5 other cases the barium was seen to pass through the sphincter, but only after prolonged delay. It is evident that, owing to individual differences, a large series would be necessary on which to base conclusions, and it is doubtful whether the values obtained by Case (250 instances of insufficiency in a series of 1,500 cases in which barium enemas were given) could be approached. There must be a fundamental difference between the response of the ileocecal sphincter to the sudden massive filling with barium and that to the gradual distention with gas and feces which occurs in cases of obstruction of the large bowel.

SIGNIFICANCE OF THE COMPETENCE OF THE ILEOCECAL SPHINCTER IN CASES OF COLONIC OBSTRUCTION

REPORTS OF CASES

The best proofs of competency of the ileocecal sphincter in cases of obstruction of the large bowel are the instances reported of gangrene and perforation of the cecum which occur in association with stenosis due to carcinoma of the sigmoid flexure. The German literature is replete with reports of cases in which the colon was greatly distended and gangrenous while the ileum was empty. Shimodaira presented 48 cases of his own and referred to the reports of Albarran and Lavillauroy, Labey, Letulle, Kocher, Kreuter, von Greyerz and others, who have mentioned cases or have referred to instances of ulceration, gangrene

or perforation of the cecum resulting from obstruction. Reichel reported 12 instances of perforation in a series of 216 cases of carcinoma of the colon. Eight of these perforations were in the cecum. Nielson reported 2 in a series of 87 cases of carcinoma of the colon. Cohen reported 2 cases. Ginzburg recently noted that "even in cases of long-standing obstruction of the colon, dilated small bowel and fluid levels are not visualized by X-ray."

I wish to report 4 illustrative cases.

CASE 1.—*History and Course.*—H. H., a white man aged 63, was admitted to the University Hospital on Aug. 30, 1931. He gave a history of gradual loss of weight for a period of six months, during which there had been attacks of pain in the abdomen. Distention and acute abdominal pain were present three days before admission, and vomiting began two days before. Examination of the abdomen at the time of admission revealed definite distention but no rigidity or tenderness either before or on rebound. Auscultation of the abdomen revealed the presence of loud borborygmi about the umbilicus and in the right lower quadrant. On rectal examination a small mass was palpated high in the left side. Roentgen examination of the abdomen showed marked distention of the large bowel with gas and the presence of levels of fluid. Because of apparent dehydration and the poor condition of the patient, it was thought best to defer operation for a few hours, during which 3,000 cc. of saline solution was administered subcutaneously. One and one-half hours before operation the patient experienced acute pain in the right lower quadrant. Examination at this time showed rigidity and tenderness both before and on rebound on the right side, neither of which signs was present at the time of admission six hours before. These findings suggested at once the presence of gangrene or perforation of the cecum. There was no obliteration of hepatic dulness on percussion.

Operation.—A large amount of turbid fluid was present in the peritoneal cavity. The cecum was enormously distended; there was no distention of the small bowel. Several rents in the cecal wall extended through the serosa and the muscularis, through which herniation of the mucosa had occurred. One of these tears measured 6 inches (15.24 cm.). The mucosa was greenish-black, suggesting gangrene extending through the entire thickness of the intestinal wall. Cecostomy was performed, and the abdomen was closed.

CASE 2.—*History and Course.*—A. H., a white man aged 73, was admitted to the University Hospital on March 25, 1932, complaining of abdominal pain, distention, vomiting and anorexia of several days' duration. He was in such poor general condition that it was impossible to ascertain the actual duration of his complaint. Examination revealed marked distention of the abdomen. Peristalsis was visible through the abdominal wall. At intervals loud borborygmi were audible, and many metallic tinkles were heard. Slight diffuse tenderness on palpation was present, but rigidity and tenderness on rebound were absent. There were no palpable masses, and the results of rectal examination were negative. The nonprotein nitrogen content of the blood was 57 mg. per hundred cubic centimeters and the chloride content, 585 mg. Roentgenograms of the abdomen revealed enormous distention of the colon. The clinical diagnosis was obstruction of the large bowel, due probably to neoplasm of the colon on the left side. Fourteen hundred cubic centimeters of a dextrose and saline solution was given subcutaneously. By gastric lavage only 50 cc. of bloodstained fluid was obtained.

Operation.—The abdomen was opened through a right McBurney incision. A markedly distended colon was presented. No distention of the small bowel was visible, the terminal portion of the ileum being collapsed. A needle connected to a mercury manometer was inserted into the colon, and the intra-intestinal pressure was determined to be 27 mm. of mercury (35 cm. of water). Eighteen hundred cubic centimeters of gas was drawn off immediately through the needle, until the recorded pressure was only 4 mm. of mercury (5 cm. of water). The needle puncture in the wall of the bowel was closed by suture. Examination revealed the presence of a signet carcinoma in the lower part of the sigmoid flexure. Sigmoidostomy was carried out. It was thought advisable to perform

Fig. 5 (case 1).—Roentgenogram of the abdomen, showing marked distention of the large bowel with gas in a case of acute obstruction of the large bowel of three days' duration, due to carcinoma of the sigmoid flexure. Distention of the cecum is the most prominent feature with no distention of the small bowel. (From Wangensteen, O. H., and Paine, J.: Treatment of Acute Intestinal Obstruction by Suction with the Duodenal Tube, J. A. M. A. **101**:1532 [Nov. 1] 1933, fig. 1.)

also a cecostomy or an appendicostomy, but the cecum could not be delivered. Therefore, ileostomy was carried out, and the abdomen was closed. The patient subsequently died.

Autopsy.—The gastro-intestinal tract presented a remarkable picture. The entire large bowel was markedly distended to a diameter of approximately 12 cm. Obstruction was due to carcinoma of the sigmoid flexure. A carcinomatous ulcer 5 cm. in diameter occurred at the point of constriction. The colon itself weighed 1,750 Gm. and contained 4,050 cc. of fluid. There were marked hypertrophy and

thickening of the wall of the entire large bowel. The ileocecal valve was competent to withstand back pressure. The small bowel was not dilated. However, there was definite evidence of hypertrophy of the wall. The hypertrophy was more evident near the ileocecal valve, the wall of the jejunum showing no signs of increased thickening.

CASE 3.—*History and Course.*—H. J., a white man aged 53, was admitted to the University Hospital on Nov. 19, 1933. The history dated from 1931, at which time he was operated on at another hospital for the relief of acute obstruction of the large bowel. Details of the operation are unknown. On admission he complained of severe, colicky, intermittent abdominal pain and complete obstipation of four days' duration. Alternating diarrhea and constipation had been present for several days prior to the onset of acute symptoms. There was increasing distention. He had vomited only once on the day before admission. Examination revealed prominent distention and both visible and audible peristalsis. A roentgenogram of the abdomen revealed marked distention of the colon. Water used in lavage of the stomach was returned clear. No gas or fluid was obtained. Nasal suction was instituted with no relief.

Operation.—Cecostomy was carried out through a right McBurney incision. A mass was palpated in the sigmoid flexure. Intra-enteric pressure (in the cecum) at the time of cecostomy was 18 mm. of mercury. Pressure was maintained at 10 mm. (13 cm. of water). The intestinal wall was edematous. There was marked congestion of the mesenteric veins. Profuse bleeding occurred from the puncture wound of the needle, indicating high venous and capillary pressures. Three hundred cubic centimeters of gas was removed by suction through the needle, and the cecum was exteriorized for cecostomy. Four hours later the pressure in the exteriorized bowel had increased to 40 mm. (52 cm. of water). The bowel was opened; a no. 24 catheter was inserted into the proximal loop, and by alternate suction and irrigation the distention was successfully reduced.

CASE 4.—*History and Course.*—Mrs. A. G., a white woman aged 72, was admitted to the University Hospital on Jan. 27, 1933, with a history of attacks of diarrhea, vomiting and abdominal pain of one month's duration. The stools had frequently been black. Obstipation, colicky abdominal pain and progressive abdominal distention had been present for three days. There had been no vomiting in the twenty-four hours prior to admission. Examination revealed marked distention of the abdomen, visible and audible peristalsis and moderate tenderness in the lower portion of the abdomen. On rectal examination a mass of questionable character was noted high in the left side, which seemed to be extrinsic to the bowel. Proctoscopic examination of the colon for 17 cm. gave negative results. Roentgenograms of the abdomen revealed distention of the large bowel only.

Operation.—Cecostomy was performed. The entire colon, especially the cecum, was distended. Exploration failed to reveal the obstructing mechanism. The intra-enteric pressure was determined to be 12 cm. of water. The postoperative course was complicated by an attack of coronary thrombosis and auricular fibrillation. On the twelfth postoperative day the temperature increased to 102 F.; the pulse became thready, and the patient complained of dyspnea, which was relieved by the administration of oxygen. No abdominal abnormalities were noted. Death occurred on the sixteenth postoperative day.

Autopsy.—Examination of the gastro-intestinal tract revealed edema of the wall of the lower portion of the ileum as one approached the ileocecal valve. There was no obstruction of the small bowel. The entire colon was moderately

dilated as far as the sigmoid flexure. The intestinal wall was much thickened and edematous. Three large perforations of the colon were evident, each approximately 4 cm. in diameter. One occurred in the transverse colon, 1 in the splenic flexure and 1 in the sigmoid flexure at the brim of the pelvis. The edges of the defects were black and appeared necrotic. The peritoneal cavity was filled with feces and showed signs of fecal peritonitis. On opening the colon the mucosa of the cecum and ascending colon was observed to be reddened and edematous. Beginning at the hepatic flexure, irregular patches of ulceration involved the mucosa and submucosa. At the splenic flexure and in the descending colon these patches fused into an almost continuous, extremely irregular area of ulceration. About 30 cm. above the anus a small adenocarcinoma had completely occluded the bowel.

Diagnosis.—The final diagnosis was carcinoma of the sigmoid flexure with intestinal obstruction and secondary ulceration and perforation of the intestinal wall and fatal peritonitis. The question of the etiology of the perforation and

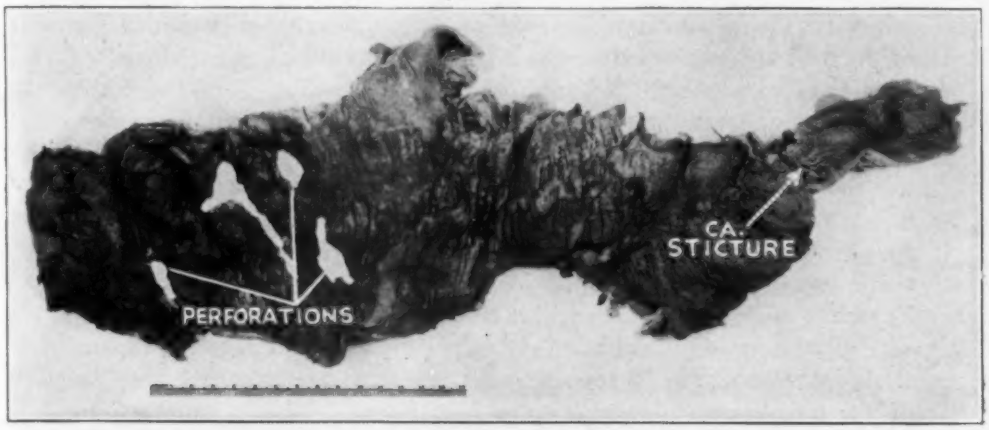


Fig. 6 (case 4).—Photograph of a postmortem specimen of the colon, showing extensive ulceration and three large perforations.

ulceration is difficult to settle. The conditions are probably explained on the basis of fecal impaction, stercoral ulcers and damage to the circulation of the wall of bowel by the increased intra-enteric pressure. It is possible that the ulceration and perforation were the result of inadequate drainage of the colon; that is, the cecostomy opening was not large enough to decompress adequately the distal part of the colon and empty it of feces. Sections of the intestinal wall studied by parasitologists did not reveal Amoeba. The diagnosis of acute ulcerative colitis on the basis of inflammation above the site of obstruction cannot be ruled out. Labey, Letulle and, most recently, Reichel have advanced this hypothesis.

EFFECT OF INCREASED INTRA-ENTERIC PRESSURE ON THE WALL OF THE BOWEL

Pathologic changes in the intestinal wall as a consequence of increased intra-enteric pressure must be secondary to the effect on the circulation of the wall of the bowel. Kocher was the first to recognize the signifi-

cance of increased intra-enteric pressure. He noted that distended incarcerated hernial loops had a congested appearance and that when gangrene appeared it was located on the antimesenteric border. He observed the so-called *Dehnungsgeschwüre* (distention-induced ulcers) and ascribed their occurrence to the effect of distention on the circulation of the wall of the bowel.

Shimodaira, a pupil of Kocher, collected 48 instances of perforation and rupture of the intestinal wall due to obstructing strictures, i. e., to carcinoma of the colon. He distended closed loops of the bowel of dogs with air until they appeared anemic. Death usually followed in three or four days. In 3 cases perforated ulcers were revealed at postmortem examination. All the loops were congested, and most presented areas of gangrene on the antimesenteric border. His explanation of the pathogenesis of these changes was that distention produces stasis in circulation, which results in thrombosis, ecchymosis, hemorrhagic infiltration of the submucosa and ulceration of the mucosa and eventually in perforation. Von Greyerz ascribed the congestion of the bowel to paralysis of the vasomotor apparatus, at first intermittent, owing to peristaltic rushes, but later constant.

Many American workers have referred to the obvious disturbance of circulation and the consequent impairment of function of the intestine as a result of increased intra-enteric pressure. Van Zwalenburg studied the effect of distention on the blood flow through the intestinal wall by means of an electric bulb within the lumen. He noted that at a pressure of 30 mm. of mercury there was capillary stasis; at 60 mm., venous stasis; at 90 mm., partial arterial arrest, and at 130 mm., complete arrest of the circulation. Gatch, Trusler and Ayers inserted cannulas into the mesenteric veins returning from closed loops and measured the volume of flow per minute under experimentally increased intra-enteric pressures. They found that for each increase of pressure there was a corresponding decrease in blood flow. Dragstedt, Lang and Millet repeated this experiment, using different levels of the gastro-intestinal tract, and found that the circulation of the duodenum was more profoundly affected than that of any other portion of the bowel. That there is disturbance of circulation in the distended bowel was confirmed recently by Jacques and his associates, who made studies of the surface temperature of the distended bowel above the site of strangulated obstruction and found the temperature to be uniformly decreased.

Van Zwalenburg attempted to explain the pathologic changes occurring in cases of intestinal obstruction on the basis of anoxia of the tissues of the intestinal wall. According to this worker, stasis of blood in the wall of the distended bowel results in a decreased supply of oxygen, which affords excellent opportunity for development of infection and its

sequelae—gangrene, necrosis and perforation. There can be no doubt that at least partial asphyxia of the wall occurs. McClure showed that the stagnant contents of obstructed loops form excellent culture mediums for growth of bacteria. The bacteria invade partially asphyxiated tissues of the intestinal wall with resulting pathologic changes. Downey showed that venous stasis is conducive to invasion by leukocytes. This would explain the infiltration of the wall of the bowel with polymorphonuclear and mononuclear cells.

Van Beuren presented a plausible explanation for the occurrence of pathologic changes in the intestinal wall in cases of obstruction. He observed that the characteristic features of the pathologic picture were areas of hemorrhagic infarction on the antimesenteric surface of the intestine and varying degrees of necrosis, culminating in perforation in cases of long-standing lesions. His explanation of the infarction of the wall of the bowel is based on purely mechanical factors. With distention the size of the bowel is increased to three times the original diameter, which results in marked thinning of the wall and in compression and decrease in the size of the lumen of the small blood vessels of the wall. The vessels elongate to accommodate the increased circumference, and this further reduces the size of their lumens. Owing to the increased intracapillary pressure and the pressure from within the bowel, the smallest vessels at the antimesenteric border rupture, producing hemorrhagic infarcts and subsequent necrosis and perforation.

EXPERIMENTAL WORK

The following experiments were undertaken to ascertain what effect constant pressures of from 30 to 50 cm. of water (present in cases of obstruction of the large bowel) have on the intestinal wall.

Dog 1.—With the animal under pentobarbital sodium anesthesia a rectal tube was inserted into the rectum of a dog and secured by a purse-string suture about the anus. Laparotomy was then performed, and a purse-string suture was placed about the lower part of the ileum. The abdomen was closed, and the colon was inflated through the rectal tube under a constant pressure of 30 cm. of water maintained for twenty-four hours by the use of the Perusse bottle. The dog was then killed. At autopsy the colon showed congested areas in the mucosa, small petechial hemorrhages in the serosa and interstitial hemorrhage in the muscle layers with infiltration of round cells in all the layers (fig. 7 *A*).

Dog 2.—The experiment was repeated, this time the colon being subjected to a pressure of 50 cm. of water. After twenty-four hours pneumoperitoneum was present. At autopsy marked congestion and areas of hemorrhagic necrosis were evident, especially in the cecum and appendix. The ileocecal sphincter was not competent to withstand this sustained pressure, and distention of the ileum had resulted in perforation through an area of hemorrhagic necrosis.

From this study it is evident that a pressure of from 30 to 50 cm. of water, if sustained for a sufficient period, will produce marked patho-

logic changes in the intestinal wall, such as are noted in clinical cases of obstruction of the large bowel. It is conceivable that lower pressures of from 10 to 20 cm. of water, if constantly maintained, could produce similar changes over a longer time.

The importance of increased intra-enteric pressure in the development of pathologic changes in the wall of the bowel might be minimized for two reasons: First, the pressures recorded in cases of experimental obstruction are comparatively low, and, second, the bowel can readily

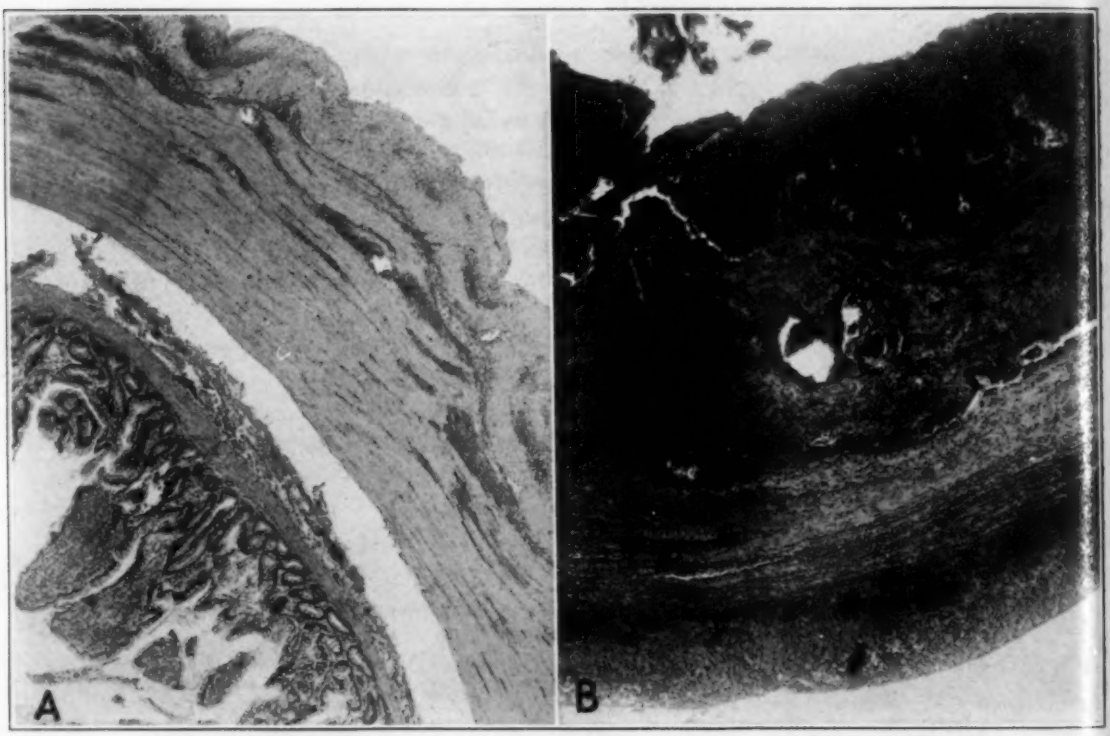


Fig. 7.—Photomicrographs of the wall of the colon (*A*) of dog 1, subjected to a pressure of 30 cm. of water for twenty-four hours, showing areas of hemorrhagic infiltration, especially in the muscularis and subserosa, and (*B*) of dog 2, subjected to a pressure of 50 cm. of water for twenty-four hours. The areas of hemorrhagic infiltration and necrosis throughout all the layers of the wall of the bowel are to be noted. The section is taken from the antimesenteric border.

accommodate itself by compensatory dilatation to slight changes in intra-enteric pressure. However, that this is not the case is illustrated by the fact that a pressure of 10 cm. of water, if constantly maintained over a sufficient period (twenty-four hours), will produce areas of hemorrhagic necrosis in closed ileal loops (unpublished thesis for the degree of Doctor of Philosophy).

There is probably a difference between the effect on the intestinal wall of sustained pressure and that of intermittent pressure, even if the intermittent pressure is high. During normal peristaltic rushes there is no doubt that high pressures obtain, but they do not damage the intestinal wall, even though temporary contraction may be sufficient to cause anemia, i. e., blanching of the wall of the bowel, because ample opportunity is afforded the intestinal wall to return to the normal state in the interval between contractions. This interval during which the wall is free from pressure is lacking, of course, when the intra-enteric pressure is sustained at a definite level. Undoubtedly, this holds true in cases of obstruction. That pathologic changes are not more often seen in the wall of the bowel is due to the ability of the bowel to relax and accommodate itself to increased intra-enteric pressure. This is especially true in cases of simple obstruction. In cases of the closed loop this phenomenon no doubt also occurs, but it is evident that at some point the sustained intra-enteric pressure must increase, bringing with it the possibility of strangulation when a sufficiently high level of constant pressure is attained.

MEASUREMENT OF THE INTRA-ENTERIC PRESSURE IN CASES OF OBSTRUCTION OF THE LARGE BOWEL

A great many observations have been recorded of the increased intra-enteric pressure in cases of experimental obstruction of the small intestine. Owings, McIntosh, Stone and Weinberg have determined experimentally that the normal sustained intra-intestinal pressure in dogs is from 2 to 4 cm. of water. In simple obstruction of the ileum they found that the sustained pressure was increased to 8 cm. of water, and during activity of the bowel it rose to as high as from 30 to 60 cm. (approximately from ten to fifteen times the normal pressure). Burget and his associates, working with closed loops of the jejunum in dogs, found pressures varying from 30 to 60 cm. of water. The average high pressure was 39.5 cm. of water (30.4 mm. of mercury). Owings and his associates found pressures of as high as 70 cm. of water in closed loops. It is evident that the pressure in closed loops attains higher levels in shorter periods than those in cases of obstruction of the simple type, in which the bowel is afforded a chance to empty itself by reversed peristalsis and regurgitation.

Morton and Sullivan compared pressures in closed loops of the jejunum with those in similar loops of the ileum. They found that the intra-enteric pressure of the jejunal loop was 7 times that which developed in ileal loops (52 cm. of water as compared to 8 cm.). They attributed this difference to the fact that secretion is much more active in the jejunum than in the ileum. It is conceivable that pressure in the colon in cases of obstruction of its distal half would rise, owing to the

fact that its contents are constantly increased by fluids and gas from the ileum, which cannot be regurgitated because of a competent ileocecal sphincter.

Stone and Firor obtained a pressure of 150 cm. of water in a case of obstruction of the lower part of the ileum of several hours' duration. So far as I can determine, this is the only clinical measurement of intra-intestinal pressure which has been recorded in a case of intestinal obstruction in man. During the past few years I have had occasion to measure the intra-enteric pressure in patients with obstruction of the large bowel who have been admitted to the University Hospital. Measurements of pressure were also made in cases of carcinoma of the large bowel or the rectum in which colostomy was performed preliminary to resection. The intra-enteric pressure was determined just prior to the release of the clamps or to making the colostomy opening. Pressures varying from 10 to 50 cm. of water were obtained.

Measurements of Intra-Enteric Pressure in Clinical Cases of Obstruction of the Large Bowel

No.	Name	Diagnosis	Procedure	Duration of Obstruction	Pressure, Cm. of Water
1	Baby B.	Imperforate anus.....	Colostomy	60 hrs.	12
2	Baby Br.	Imperforate anus.....	Colostomy	18 hrs.	10-26
3	R. D.	Carcinoma of the rectum..	Colostomy	60 hrs.	12
4	A. S.	Carcinoma of the rectum..	Colostomy	72 hrs.	18
5	E. G.	Carcinoma of the rectum..	Colostomy	104 hrs.	12-16
6	E. C.	Carcinoma of the rectum..	Colostomy	80 hrs.	13
7	C. A.	Carcinoma of the sigmoid flexure and acute obstruction	Cecostomy	8 days	23-26
8	A. H.	Carcinoma of the sigmoid flexure with obstruction	Cecostomy	?	35
9	J. C.	Carcinoma of the ascending colon with obstruction	Cecostomy	4 days	24
10	T. W.	Carcinoma of the rectum with obstruction	Colostomy	3 days	15
11	H. J.	Carcinoma of the sigmoid flexure with obstruction	Cecostomy	3 days	52
12	A. G.	Carcinoma of the sigmoid flexure with obstruction	Cecostomy	3 days	12

Six of the determinations of pressure noted in the table were made in cases of acute obstruction of the large bowel. In 4 cases obstruction was due to carcinoma of the sigmoid flexure; in 1, to carcinoma of the ascending colon, and in 1, to carcinoma of the rectum. The pressures were measured at the time of operation. In 4 instances of carcinoma of the rectum among the remaining cases colostomy was carried out as a procedure preliminary to resection. The colostomy opening was made only after definite clinical signs of obstruction (distention and colicky pain) became evident. In some of these cases roentgen examination preliminary to opening the bowel revealed only distention of the large

bowel. It was noted that in the cases in which nasal suction was instituted after operation the patients were more comfortable and were able to tolerate the obstruction for a somewhat longer period. However, in spite of suction definite distention of the large bowel developed in all the cases, owing no doubt to the fact that gas and chyme could pass into the cecum but could not be sucked out because of the competency of the ileocecal sphincter. In 2 cases colostomy was performed for the correction of imperforate anus in a child. The intra-enteric pressure was measured when signs of obstruction necessitated making the colostomy opening.

It is to be noted that all these pressures are well below that employed by the roentgenologist to force the ileocecal valve. They represent the level of sustained pressure within the bowel. No doubt, higher pressures would be recorded during peristaltic rushes. It is regrettable that no determinations of pressure were made in the case of gangrene of the cecum which is recorded as the first in this series.

COMMENT

It has been shown that the ileocecal sphincter is competent to withstand pressures within physiologic limits (pressures of from 10 to 50 cm. of water) which conceivably might occur in the course of obstruction of the large bowel. A competent ileocecal sphincter at once converts a simple type of obstruction into a closed loop with all the inherent dangers of strangulation due to increased intra-enteric pressure. Experimentally, the effect of such sustained pressures is shown by the development of areas of hemorrhagic necrosis in the colon of dogs. That similar changes occur in the human colon is evident from a perusal of the literature cited and from the reports of cases in this paper.

The term "ileocecal valve" is a misnomer; the organ is more rightly called the ileocecal sphincter. It is subject to definite nervous control, and its competency depends on the tonicity of the fibers of the sphincter. That the tone of the sphincter is increased by stimulation of the sympathetic nerves is confirmed by the experiments in this study. Also, it has been shown that stimulation of the distal part of the colon increased the resistance of the sphincter to back pressure to approximately three times that of the normal sphincter. Stimulation of the parietal peritoneum, the stomach or the small bowel had no such effect. It is conceivable that the resistance of the ileocecal sphincter to back pressure is greatly increased in cases of intrinsic pathologic conditions of the colon. Stimulation of the distal portion of the colon, acting through Auerbach's plexus, increases the tone of the ileocecal sphincter, making it more competent.

Several important clinical observations present themselves with relation to a competent ileocecal sphincter in cases of obstruction of the large bowel.

1. Vomiting is a late symptom in cases of obstruction of the large bowel. The competent ileocecal valve allows material to pass into the colon but none to be regurgitated into the small bowel and stomach. Pain, distention and obstipation may be present for several days before the onset of vomiting. Vomiting may then be due to reflexes set up by distention of the colon (distention of any hollow viscus may produce vomiting). It is interesting to note that in the cases cited aspiration of the stomach resulted in the return of only a few cubic centimeters, in spite of the fact that these cases represented late stages of obstruction.

2. Nasal suction as a method of decompression is of little value in the treatment of acute obstruction of the large bowel with considerable distention.

In instances of obstruction of the colon on the left side in which the distention has not been great, the employment of nasal suction will frequently permit one to attack the obstruction directly after subsidence of the obstructive features. The virtue of decompression by the duodenal tube in these cases lies essentially in the fact that aspiration of swallowed air and fluid from the upper portion of the intestinal canal and stomach prevents increase in the distention. As already noted, when the method is employed in this manner in cases of postoperative obstruction the appearance of obstructive symptoms and distention was delayed, allowing one to defer making the colostomy opening for several days.

3. A single roentgenogram of the abdomen of a patient with clinical intestinal obstruction should differentiate between obstruction of the small bowel and that of the large. Roentgenographic findings of a markedly distended colon and dilatation of the cecum with no visible loops of small bowel should clinch the diagnosis of obstruction of the sigmoid flexure. Roentgenography is the only accurate method of determining the degree of distention and the segment of bowel involved.

4. All acute obstructions of the large bowel exhibiting considerable distention should be treated as obstructions of the closed loop type with potential strangulation, by means of operative decompression (cecostomy). Simple mechanical obstruction of the small bowel has been treated successfully at the University Hospital by decompression by means of nasal suction (Wangensteen). This method, of course, is impossible in cases of the variety found in the large bowel, owing to the presence of a competent ileocecal sphincter. Adequate decompression by cecostomy should be carried out in cases of great distention to prevent, if possible, the complications resulting from continuous

increased intra-enteric pressure, i. e., gangrene and perforation of the intestinal wall. Patients who present only partial or low grade obstruction of the colon can be prepared for operation by medical management, and further distention of the colon can be prevented by the use of siphonage by nasal suction.

CONCLUSIONS

The ileocecal sphincter is competent to withstand moderate increases of intra-enteric pressure, such as occur in cases of obstruction of the large bowel.

Obstruction of the colon with considerable distention is of the closed loop type, because the presence of a competent ileocecal sphincter effectually prevents regurgitation into the ileum.

The intra-enteric pressure which obtains in cases of obstruction of the large bowel has been measured in clinical cases and has been found to vary from 10 to 50 cm. of water.

When the colon of the dog is exposed to a constant pressure of from 30 to 50 cm. of water for a sufficient period (twenty-four hours) areas of gangrene and hemorrhagic necrosis appear in the wall of the bowel.

The clinical cases cited illustrate some of the aforementioned facts.

BIBLIOGRAPHY

Albarran, J., and Lavillauroy: Ulcérations intestinales dans l'occlusion intestinale chronique, Bull. Soc. anat. de Paris **73**:61, 1898.

Alvarez, W. C.: The Mechanics of the Digestive Tract, New York, Paul B. Hoeber, Inc., 1929, p. 227.

Anschütz, W.: Ueber den Verlauf des Ileus beim Darmkarzinom und den lokalen Meteorismus des Coecum bei tiefsitzenden Dickdarmverschluss, Arch. f. klin. Chir. **68**:195, 1902.

Balli, R.: Sphincters of the Colon, Radiology **12**:484, 1929.

Bauhinus, C.: Theatrum anatomicum, Frankfort-on-Main, Matthaei Beckeri, quoted by Walcker.

Blamoutier, P.: Les mouvements antiperistaliques du gros intestin, Paris méd. **1**:325, 1925.

Burget, G. E.; Martzloff, K.; Suckow, G., and Thornton, R. C. B.: Closed Intestinal Loop: Relation of Intraloop (Jejunum) Pressure to the Clinical Condition of the Animal, Arch. Surg. **21**:829 (Nov.) 1930.

Burt, C. A. V.: Pneumatic Rupture of the Intestinal Canal with Experimental Data Showing the Mechanism of Perforation and the Pressure Required, Arch. Surg. **22**:875 (June) 1931.

Cannon, W. B.: The Movements of the Intestine Studied by Means of the Roentgen Rays, Am. J. Physiol. **6**:251, 1902.

Relation of Tonus to Antiperistalsis in the Colon, Am. J. Physiol. **29**:238, 1911.

Carman, R. D., and Miller, A.: The Roentgen Diagnosis of Diseases of the Alimentary Tract, ed. 2, revised, Philadelphia, W. B. Saunders Company, 1921.

Case, J. T.: X-Ray Observations on Colonic Peristalsis and Antiperistalsis with Special Reference to the Function of the Ileocolic Valve, M. Rec. **85**:415, 1914.

Cohen, Moritz: Caecumperforation bei tiefsitzendem Dickdarmverschluss, Deutsche med. Wchnschr. **25**:1267, 1914.

Cole, L. G.: Ileo-Cecal Incompetency, M. Clin. North America **1**:689, 1917.

Dale, H. H.: On Some Physiological Activities of Ergot, J. Physiol. **34**:179, 1906.

Dauriac, J.: Note sur l'irrigation totale et antiseptique du tube digestif, Progrès méd. **18**:217, 1893.

Dietlen, Hans: Die Insuffizienz der Valvula ileocecalis im Röntgenbild, Fortschr. a. d. Geb. d. Röntgenstrahlen **21**:23, 1914.

Downey, H.: Reaction of Blood and Tissue Cells to Acid Colloidal Dyes Under Experimental Conditions, Anat. Rec. **12**:429, 1917.

Dragstedt, C. A.; Lang, V. G., and Millet, R. F.: The Relative Effects of Distention on Different Portions of the Intestine, Arch. Surg. **18**:2257 (June) 1929.

Drummond, Hamilton: Observations on the Functions of the Colon with Special Reference to the Movement of Enemata, Brit. M. J. **1**:245, 1914.

Elliot, T. R.: On the Innervation of the Ileo-Colic Sphincter, J. Physiol. **31**:157, 1904.

——and Barclay, Smith E.: Antiperistalsis and Other Muscular Activities of the Colon, J. Physiol. **31**:272, 1904.

Fabricus ab Aquapendente, H.: Opera omnia anatomica et physiologica Lipsiae, Leipzig, J. F. Gleclitsch, 1687; cited by Plesch.

Gatch, W. D.; Trusler, H. M., and Ayers, K. D.: Effect of Gaseous Distention on Obstructed Bowel: Incarceration of Intestine by Gas Traps, Arch. Surg. **14**:1215 (June) 1927.

von Genersich, A.: Le lavage du canal digestif, Progrès méd. **21**:38, 1893.

Ginzburg, L.: X-Ray Diagnosis of Acute Intestinal Obstruction Without Use of Contrast Media, Ann. Surg. **96**:368, 1932.

von Greyerz, W.: Ueber die Oberhalb von Dickdarmverengerungen auftretenden Darmgeschwüren, Deutsche Ztschr. f. Chir. **77**:57, 1905.

Groedel, F.: Die Insuffizienz der Valvula ileocecalis im Röntgenbild, Fortschr. a. d. Geb. d. Röntgenstrahlen **20**:162, 1913.

Hammer, Gerhard: Untersuchungen über die Function der Valvula Bauhini, Arch. f. klin. Med. **157**:1, 1927.

Heile, B.: Beobachtungen über die Resorption im Dünn- und Dickdarm, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **14**:474, 1905.

Die Erkennung und Behandlung von Bauhinospasmus (Spasmus des Sphincter ileocolicus), Zentralbl. f. Chir. **48**(pt.1):506, 1921.

Herlyn, K. E.: Zur Darstellung der Ileozökalklappe im Röntgenbild, Fortschr. a. d. Geb. d. Röntgenstrahlen **47**:644, 1933.

Hinrichsen, J., and Ivy, A. C.: Studies on the Ileo-Cecal Sphincter of the Dog, Am. J. Physiol. **96**:494, 1931.

Hromada, G.: Zur Insufficienz der Valvula Bauhini, Arch. f. klin. Chir. **117**: 784, 1921.

Jacques, L.; Droegmueller, W. A., and Buchbinder, J. R.: Viability of Strangulated Loops: Experimental Study, Surg., Gynec. & Obst. **55**:559, 1932.

Joltrain, E.; Baufle, P., and Coope, R.: Essai de mesure de la pression du gros intestin—ses variations: Application à la clinique, Bull. et mém. Soc. méd. d. hôp. de Paris **43**:211, 1919.

Kocher, T.: Ueber Ileus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **4**:195, 1899.

Kreuter, E.: Dehnungsgangräne des Coecum bei Achsendrehung der Flexura sigmoidea und bei Abknickung des Blinddarms, Arch. f. klin. Chir. **70**:518, 1903.

Labey: Ulcérations dysentériformes du gros intestin dans un cas de rétrécissement cancereux du rectum ayant déterminé de l'occlusion chronique, Bull. Soc. anat. de Paris **73**:130, 1898.

Larson, L. M., and Bargen, A. J.: Physiology of the Colon, Arch. Surg. **27**:1 (July) 1933.

Letulle, M.: Rupture du colon ascendant par diastasis, examen microscopique, Bull. Soc. anat. de Paris **74**:588, 1899.

Morton, J. J., and Sullivan, W. C.: Comparison Between Simultaneous Equal-Sized Closed Obstructions of Duodenum and Ileum, Arch. Surg. **21**:531 (Sept.) 1930.

McClure, R. D.: An Experimental Study of Intestinal Obstruction, J. A. M. A. **49**:1003 (Sept. 21) 1907.

Nielson, Michael: Om tarmperforationer ved cancer coli, Bibliot. f. læger **119**: 235, 1927.

Owings, J. C.; McIntosh, C. A.; Stone, H. B., and Weinberg, J. A.: Intra-Intestinal Pressure in Obstruction, Arch. Surg. **17**:507 (Sept.) 1928.

Piccolhomini, Archangelus: Anatomiae praelectiones, 11, Rome, Italy, Lectura, B. Bonfadini, 1586; cited by Plesch.

Plesch, J.: Regurgitatio coloiliaca, Ztschr. f. klin. Med. **108**:340, 1928.

Reichel, Paul: Klinischer Teil, in Reichel, P., and Staemmler, M.: Die Neubildungen des Darmes, Neue deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1933, vol. 33, p. 243.

Rigler, Leo: Personal communication.

Rost, Franz: The Pathological Physiology of Surgical Diseases, Philadelphia, P. Blakiston's Son & Co., 1923, p. 199.

Rutherford, A. H.: The Ileo-Cecal Valve, New York, Paul B. Hoeber, Inc., 1914.

Schwartz, Gottwald: Zur Physiologie und Pathologie der menschlichen Dickdarmbewegungen, München. med. Wchnschr. **28**:1489, 1911.

Senn, Nicholas: Experimental Surgery, Chicago, Chicago Medical Book Company, 1889, p. 476.

Shimodaira, Yosai: Experimental Untersuchungen über die Entstehung von sogenannten Dehnungsgeschwüren des Darmes, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **22**:229, 1911.

Short, Rendel: Observations on the Ileo-Cecal Valve in Man, Brit. M. J. **2**:164, 1919.

Sobotta, J., and McMurrich, J. P.: Atlas and Text-Book of Human Anatomy, Philadelphia, W. B. Saunders Company, 1914.

Stone, N. E., and Firor, W.: Absorption in Intestinal Obstruction; Intra-Intestinal Pressure as a Factor, Tr. South. S. A. **37**:173, 1924.

Tönnis, W.: Die Funktion der Valvula ileocoecalis, Arch. f. d. ges. Physiol. **204**: 477, 1924.

——and Sichler, P.: Zur Diagnose der Insuffizienz der Valvula ileocecalis, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **40**:648, 1928.

Van Beuren, F. T., Jr.: Relation Between Intestinal Damage and Delayed Operation in Acute Mechanical Ileus, Ann. Surg. **72**:610, 1920.

Van Zwalenburg, C.: Strangulation Resulting from Distention of Hollow Viscera, Ann. Surg. **46**:780, 1907.

Hydraulic Vicious Circle as It Develops in Acute Appendicitis, Am. J. Surg. **16**:427, 1932.

Walcker, F. I.: Die Bedeutung der Bauhinischen und appendikulären Klappe: Experimentelle und anatomische Untersuchungen, Arch. f. klin. Chir. **170**: 706, 1932.

Wangensteen, O. H.: Early Diagnosis of Acute Intestinal Obstruction with Comments on Pathology and Treatment, with Report of a Successful Decompression of Three Cases of Mechanical Bowel Obstruction by Nasal Catheter Suction Siphonage, West. J. Surg. **40**:1, 1932.

von Ziemssen, H.: Die künstliche Gasaufblähung des Dickdarmes zur diagnostischen und therapeutischen Zwecken, Deutsches Arch. f. klin. Med. **33**:235, 1883.

MARCH FOOT ASSOCIATED WITH UNDESCRIBED CHANGES OF THE INTERNAL CUNEIFORM AND METATARSAL BONES

I. H. MASERITZ, M.D.
BALTIMORE

For many years march foot was closely linked with the soldier, and the earlier literature was derived, almost in its entirety, from cases which had been seen in the ranks of the German and French armies. It is by no means a condition confined entirely to the soldier, but one that is seen less frequently in civilian life. For some years comparatively little attention was given to the subject; almost nothing was written, and it literally passed into obscurity. It is not surprising that many physicians today possess little or no knowledge of the condition, and it is more than likely that numerous cases have escaped recognition. In recent years Jansen [1] and others have again brought this subject to light. Undoubtedly interest has been stimulated, and much more should now be added to the chapter on marching fractures.

The primary purpose of this paper is to deal with several undescribed changes in march foot, but I feel that in conjunction with this a thorough discussion of the subject will not be out of place.

Breithaupt [2] described a swelling of the feet that commonly occurred in soldiers after a long march. He named this condition *Fussgeschwulst* and attributed it to an inflammation of the tendon sheaths. Weisbach [3] did not agree with the explanation of Breithaupt but felt that the changes responsible were those of an inflammation involving the ligaments and therefore applied the term syndesmitis metatarsea. Pauzat [4] described the condition as an edematous swelling of the feet, apparent on the dorsal and plantar aspects, and consisting of two stages, the first a simple soft tissue swelling and the second a periosteal proliferation. He further wrote that the second, third and fourth metatarsal bones were

---

From the Department of Orthopedic Surgery, University of Maryland Medical School, and Mercy Hospital.

1. Jansen, Murk: Die Fussgeschwulst und ihre Ursache, Ztschr. f. orthop. Chir. **35**:8, 1916; March Foot, J. Bone & Joint Surg. **8**:262 (April) 1926.

2. Breithaupt: Zur Pathologie des menschlichen Fusses, Med.-Ztg. **24**:169 and 175, 1855.

3. Weisbach: Die sogenannte Fussgeschwulst (Syndesmitis metatarsea) des Infanteristen, Deutsche mil.-ärztl. Ztschr. **6**:551, 1877.

4. Pauzat, J. E.: De la périostite ostéoplastique des métatarsiens a la suite des marches, Arch. de méd. et pharm. mil. **10**:337, 1887.

found to be tender and were the only bones that ever displayed changes. He considered these changes to be the result of an osteoplastic periostitis. Poulet [5] viewed the condition as a rheumatic osteoperiostitis. Martin [6] reported three types, one which was a simple periostitis, a second in which tendonitis alone existed, and a third that was described as a periostitis complicating mediotarsal arthritis. Busquet,[7] on the other hand, thought that there were three distinct groups of *Fussgeschwulst*: (1) a direct periostitis, (2) an indirect periostitis and (3) a diathetic periostitis.

With the advent of the x-ray, Stechow [8] described the first cases of march foot that were studied roentgenologically. He demonstrated that march foot not only showed involvement of the periosteum but very frequently fractures, and he presented 34 cases with a single and 1 case with a double fracture. The frequency of involvement was as follows: first metatarsal bone, left foot, 0, and right foot, 0; second, left foot, 1, and right foot, 0; third, left foot, 11, and right foot, 8; fourth, left foot, 8, and right foot, 6; fifth, left foot, 0, and right foot, 0.

Schulte [9] reported 53 cases of *Fussgeschwulst*, which involved the second metatarsal bone in 33 instances, the third in 18 and the fourth in 2. The first and fifth metatarsal bones were never involved, and he considered each case one of fracture. One of his descriptions of a soldier with march foot gives the characteristic clinical picture. The soldier, after a long march, returned to camp complaining of a burning sensation in the midportion of his right foot. On the following morning, in spite of the fact that his foot had become swollen, he undertook an hour's march. The pain and swelling increased. Schulte mentioned that there was a distinct point of maximum tenderness over the proximal half of the second metatarsal bone with no tenderness over the first and fifth metatarsal bones. Twenty days later he was able to feel a firm lump in the area corresponding to the previous point of maximum tenderness, and the mass, by x-ray film, proved to be bony callus and a fracture. As to cause, Schulte mentioned that in jumping, running,

---

5. Poulet, A.: De l'ostéopériostite rhumatismale des métatarsiens, Arch. de méd. et pharm. mil. **12**:245, 1888.

6. Martin, A.: Inflammation périosto-arthritique du pied á la suite des marches, Arch. de méd. et pharm. mil. **18**:336, 1891.

7. Busquet, P.: De l'ostéopériostite rhumatismale des métatarsiens, Rev. de chir. Paris **17**:1065, 1897.

8. Stechow: Fussödem und Roentgenstrahlen, Deutsche mil.-ärztl. Ztschr., **26**:465, 1897; Fussödem und Roentgenstrahlen, Internat. Cong. f. Hyg. & Demogr. **8**:99, 1898.

9. Schulte: Die sogenannte Fussgeschwulst, Arch. f. klin. Chir. **55**:872, 1897.

long marches or even walking while on nonactive duty the men would experience sudden attacks of pain and present typical march foot.

Kirchner [10] reported a total of 82 cases, among which those involving the second and third metatarsal bones made up by far the largest percentage. He mentioned that the condition was common in civilian life, and his series included bakers, painters, laborers and soldiers. He further mentioned that each case was one of fracture and that trauma was the direct predisposing factor. The site of involvement was given as follows: second metatarsal bone, 40 cases; third, 31; fourth, 1; fifth, 1; second and third, 4; third and fourth, 5.

Thiele,[11] on the other hand, with a total of 33 cases, reported fractures in only 14 instances, but stated that periostitis was present in all, and that crepitation was elicited in 7 and abnormal mobility in 5. Periosteal proliferation could be detected in each case after a period of from eight to ten days.

Blecher [12] added reports of 78 cases to the literature. He was the first to make mention of recurrences in old fractures and reported 5 cases in which fresh fractures occurred in old healed ones a second, and in 2 cases a third, time. Definite fractures were observed in 30 cases, periostitis alone in 29 and no changes in 19. As to etiology, he felt that two factors were present: (1) an overburdening of soldiers, causing an undue strain on the metatarsal bones, and (2) a direct mechanical force causing the fracture.

Meiser [13] offered a point of some significance in stating that a fracture, although present in every typical case of march foot, could be demonstrated in only one third of the instances. Another was his theory of the etiology of march foot. He wrote that an elongated metatarsal bone is the probable cause of a weak foot, and that the weakness of the foot is proportional to the elongation of the metatarsal bone. (Dodd [14] in a more recent article considers the short first metatarsal bone as an outstanding factor in the cause of march foot.)

---

10. Kirchner, A.: Ueber das Wesen der sogenannten Fussgeschwulst, Wiesbaden, J. F. Bergmann, 1898; Die Fussgeschwulst, Deutsche mil.-ärztl. Ztschr. **28**:79, 1899; Die Aetiologie der indirekten Metatarsalfraktur, Arch. f. klin. Chir. **77**:241, 1905.

11. Thiele: Ueber Frakturen der Metatarsalknochen durch indirekte Gewalt (Fussgeschwulst), Deutsche med. Wchnschr. **25**:158, 1899; Weiterer Beitrag nach der Ursache der sogenannten Fussgeschwulst, Deutsche mil.-ärztl. Ztschr. **29**:129, 1900.

12. Blecher: Fussgeschwulst, Knochenbruch und Knochenhautentzündung, Deutsche mil.-ärztl. Ztschr. **31**:321, 1902; Entstehung der Fussgeschwulst, ibid. **32**:3, 1903.

13. Meiser: Die Brüche der Mittelfussknochen als Ursache der Fuss- oder Marschgeschwulst, Fortschr. a. d. Geb. d. Röntgenstrahlen **4**:105, 1901.

14. Dodd, H.: Pied Fourcé or March Foot, Brit. J. Surg. **21**:131 (July) 1933.

In direct contrast to Meiser, Thalwitzer [15] was a supporter of the theory that march foot is caused by periostitis and felt that fractures were present only when demonstrable.

Eyles' [16] dissertation conforms more to the general than to some of the individual concepts of march foot. He offered one or more of the following points as positive findings in a typical case: (1) a definite area of callus, (2) a complete fracture without displacement, (3) a complete fracture with displacement, (4) an incomplete fracture and (5) periosteal thickening of the shaft of one or more bones.

In his experience the second and third metatarsal bones were most commonly involved and usually in the distal third.

Tobold [17] offered 1,500 cases with details of statistical value. Changes were found in 67.3 per cent of all cases. Fractures occurred in 49.7 per cent and periostitis in 11.9 per cent. Old fractures were found in 5.7 per cent and no fractures in 32.7 per cent of the cases. The metatarsal bones were the seat of involvement in the following percentages: second left, 13.2; second right, 10.8; third left, 10.3; third right, 8.7.

Nion [18] also contributed statistics of value. In a series of 575 cases which he examined at the Roentgen Institute, he found changes in 330. The left foot was involved in 118 instances; the right, in 115 (including double changes). Fractures were seen in the metatarsal bones in 228 cases as follows: in the second metatarsal bone in 112; in the third in 98; in the fourth in 17; in the first in 1.

Runström [19] in an article on fresh subperiosteal hemorrhage in fractures of the metatarsal bones brought to light some interesting and important facts. To quote him verbatim:

> My material at the Military Hospital comprises partly injuries after definite traumas—direct and indirect—and partly "marching injuries" without trauma . . . I have divided the material into three groups according to the roentgenologic observations:
>
> Group 1. Fourteen cases show a fracture or a fine fissure without dislocation and a thin spindle-shaped deposit. Two of these cases have occurred after a definite trauma and have been examined by means of the roentgen rays on the fourth and sixth days. Of the twelve remaining cases eight have been examined on the second to the fourth day and the rest on the sixth to the eighth day after the trauma.

---

15. Thalwitzer: Zur Aetiologie der Fussgeschwulst, Deutsche mil.-ärztl. Ztschr. **31**:435, 1902.

16. Eyles, F.: Die Marschfraktur, München. med. Wchnschr. **62**:1703, 1915.

17. Tobold: Zur Kasuistik der Mittelfussknochenbrüche, Deutsche mil-ärztl. Ztschr. **32**:573, 1903.

18. Nion: Zur Statistik der Mittelfussknochenbrüche, Deutsche mil.-ärztl. Ztschr. **32**:200, 1903.

19. Runström, Gösta: Roentgen Picture of Fresh Subperiosteal Hemorrhages in Fractures of the Metatarsals, Acta radiol. **3**:42, 1924.

Group. 2. Twelve cases show a thin spindle-shaped deposit but no absolutely provable fracture. Three of them have occurred after a definite trauma and have been examined on the first, fifth and seventh days. Of the "marching injuries" six have been examined on the third to the sixth day and the other ones on the seventh to the eighth day.

Group 3. Nineteen cases show fractures with distinct dislocation but no spindle-shaped deposit. Six of these have occurred after a definite trauma and have been examined on the first to the fifth day. Of the "marching injuries," twelve have been examined on the first to the fifth day, and one on the eighth day.

He concluded that a fracture is present in every instance and that the spindle-shaped deposit is merely subperiosteal hemorrhage, which cannot be present, and is not present, in group 3, in which dislocation of the fragments has occurred.

Jansen in 1916, and again in 1926, offered a more radical concept of the etiology of march foot. He felt that march foot is a complication of a subacute weakness of the foot and that the interosseus muscle plays an important rôle in bringing about bone changes. He emphasized the origin of the interossei, showing that they arise on the sides of the metatarsal shafts with the exception of the first bone and the lateral aspect of the fifth bone. He stated further that the spasm of the muscle bellies gives rise to an edema and hypertrophy of the muscle tissues and periosteum. At times an acute subperiosteal hemorrhage is formed. The periosteal changes are never on the outer border of the fifth or either border of the first metatarsal bones, only because the muscle does not have any of its origin in these areas. The bone, possessing no expansibility, becomes partly absorbed, brittle and more susceptible to fracture.

Dodd [14] questioned Jansen's theory of vascular disturbances and bone absorption and felt that the various changes as suggested by different authors—namely, tenosynovitis, spasm of muscles, arthritis, periostitis, synovitis—are merely manifestations of the weak foot. He emphasized the four points of Morton which are commonly seen in weak feet and offered a study of 14 cases which corroborated his theory.

Morton's points as given by Dodd are:

1. Laxity of the joint between the internal cuneiform bones and between them and the scaphoid, resulting in hypermobility of the first metatarsal. This is shown by a distinct separation between the first and second cuneiforms, resembling a backward continuation of the first intermetatarsal space.

2. Shortness of the first metatarsal, this causing over-pronation of the foot, i. e., planus of the anterior part of the foot, as the head of the metatarsal is not long enough to reach the ground in the normal attitude.

3. Posteriorly located sesamoid bones at the head of the first metatarsal, these representing its point of contact with the ground, and when they are posterior to the line of the head of the second metatarsal, they have practically the same effect as that of a short metatarsal, i. e., they prevent adequate weight-bearing by the first metatarsal.

4. Morton points out a further acquired physiological characteristic resulting from these defects; it is an enlargement of the shaft of the second metatarsal bone, especially in its transverse diameter, arising in response to the increased burden thrown on it by the incompetent first metatarsal.

Dodd added a fifth factor in march foot, a thickening of the outer border of the first metatarsal bone, a finding in direct contrast to that of Jansen, who stated that periosteal proliferation is never present in this metatarsal bone. Dodd, as did previous writers, stated that the

Fig. 1.—Left foot, anterior-posterior view, on Aug. 29, 1934, showing a recent fracture at the base of the fifth metatarsal bone and an old fracture in the proximal fourth of the second metatarsal shaft (indicated by arrows). On the medial aspect of the middle third of the third metatarsal bone periosteal thickening may be seen. The internal cuneiform bone presents slight lipping. (Note that the heads and necks of the metatarsal bones are normal.)

first metatarsal bone, being thicker and stronger than the remaining ones, is forced to bear the greater brunt of the body's weight, but that following a relaxation of ligaments the second, then the third and remaining metatarsal bones take on the strain but because of size are unable to sustain the force and fracture.

PATHOLOGY OF MARCH FOOT

Dodd reported a case in which a Lisfranc amputation was performed because of a mistaken preoperative diagnosis of sarcoma. The case proved to be one of march foot. The pathologic specimen was reported to show rarefying osteitis of the metatarsal bone associated with chronic inflammatory periostitis.[20]

Strauss[21] reported a case of march foot that was preoperatively diagnosed as neoplasm and, because of this, the entire metatarsal bone

Fig. 2.—The same foot as in figure 1, lateral view.

was removed. Pathologically the condition was found to be "a fracture of the shaft of the bone with incomplete repair, small hemorrhage and exuberant callus."

---

20. Deutschländer (Ueber entzündliche Mittelfussgeschwulste, Arch. f. klin. Chir. **118**:530, 1921) considered march foot to be an inflammatory periostitis.

21. Straus, F. H.: Marching Fractures of Metatarsal Bones with a Report of the Pathology, Surg., Gynec. & Obst. **54**:581 (March) 1932.

Each remaining author [22] has added to the long list of contributions to the knowledge of march foot, some by reports of cases, others by presentations of theory, and still others by articles of general discussion. Theories are numerous and probable causes many. Details were abstracted only when considered to be of some significance. Finer points, however, were very often lacking. At the close of this bibliographic abstract one important point should be emphasized, namely, that of the usual site of fracture or involvement in march foot. The proximal, middle and distal thirds were the only areas of bony changes suggestive of fracture, and these were usually in the middle and proximal thirds, never in the heads or bases of the bones. The tarsal bones, too, never showed any changes whereas the phalanges suggestively did in several instances.[23]

---

22. Bähr, F.: Die Fussgeschwulst und ihre Beziehungen zum vorderen Frontalgewölbe, Ztschr. f. Orthop. Chir. **32**:310, 1913. Beely, F.: Zur Mechanik des Stehens: Ueber die Bedeutung des Fussgewölbes, Arch. f. klin. Chir. **27**:457, 1882. Boisson, A., and Chapotot, E.: Le pied forcé: Etude sur la nature et la pathogénie des lésions de l'avant-pied provoquées par la marche, Arch. de méd. et pharm. mil. **33**:81, 1899. Goldman, S., E.: March Foot with Fracture of Metatarsal Bone, J. Bone & Joint Surg. **10**:228 (April) 1928. Hamann, H.: Ueber subcutane Fracturen der Metacarpal- und Metatarsalknochen mit besonderer Berücksichtigung des sogenannten militärischen Fussödems, Dissert., Greifswald, J. Abel, 1902. de Holstein, V.: Les fractures des métatarsiens, provoquées par la marche, Semaine méd. **19**:187, 1899. Marey and Demeny: Locomotion humaine; mécanisme du saut, Compt. rend. Acad. d. sc. **101**:489, 1885. Messerer, O.: Ueber die Elasticität und Festigkeit der menschlichen Knochen, Stuttgart, J. G. Cotta, 1880. von Meyer, G. H.: Die Statik und Mechanik des menschlichen Knochengerüstes, Leipzig, Wilhelm Engelmann, 1873; Statik und Mechanik des menschlichen Fusses, Jena, Gustav Fischer, 1886. Momburg: Die Entstehungsursache der Fussgeschwulst, Deutsche Ztschr. f. Chir. **73**:425, 1904. Monteith, W. B. R.: Case of March Foot (Pied Forcé) with Signs of Old and Recent Injury, Brit. J. Surg. **21**:708 (April) 1934. Muskat, Gustav: Die Brüche der Mittelfussknochen in ihrer Bedeutung für die Lehre von der Statik des Fusses, Samml. klin. Vortr. n.F. October 1899, no. 258 (Chir., no. 76), p. 1387. Nanoury, G.: De la fracture du métatarse chez les jeunes soldats, Rev. d'orthop. **9**:349, 1898. Nasse and Borchardt, in von Bergmann, E.; von Bruns, P., and Mikulicz, J.: Handbuch der praktischen Chirurgie, ed. 3, Stuttgart, Ferdinand Enke, 1907, p. 947. Nimier, H.: De l'entorse métatarsienne chez les fantassins, Arch. de méd. et de pharm. mil. **21**:61, 1893. Pirie, A. H.: Marching Fractures, Lancet **2**:47 (July 14) 1917. Rittershausen: Zur Frage der Fussgeschwulst, Deutsche mil.-ärztl. Ztschr. **28**:18, 1899. Schipmann, G.: Zur Kasuistik der Brüche der Metatarsalknochen, Deutsche med. Wchnschr. **25**:319, 1899. Sleeswijk, R.: Warum kommt die Fussgeschwulst beim Militär am meisten vor? Deutsche mil.-ärztl. Ztschr. **29**:552, 1900. Speed, J. S., and Blake, T. H.: March Foot, J. Bone & Joint Surg. **15**:372 (April) 1933. Tiedemann, E.: Ueber die Häufigkeit und das Wesen der Fussgeschwulst, Dissert., Berlin, O. Francke, 1904. Trnka: Die subcutanen Frakturen der Metatarsalknochen, Wien. med. Wchnschr. **49**:1879, 1899.

23. Dodd [14] interpretated phalangeal changes as those consisting of thickenings and fracture.

Retrospectively it may be stated that march foot is a complication of the "strained" foot, characterized usually by a sudden onset of pain and swelling on the dorsum and, to some degree, on the plantar aspect of the middle part and forepart of the foot. Clinically there is presented a tenderness over the shafts of one or more metatarsal bones and commonly at the junction of the middle and distal thirds of the second and

Fig. 3.—The anterior-posterior and lateral views of the left foot taken on Nov. 30, 1934. There are fractures of the head of the second and neck of the third metatarsal bone (indicated by arrows). The internal cuneiform bone is fragmented in part (arrows). The fracture at the base of the fifth metatarsal bone is almost completely healed. (Note the unusual amount of callus in the neck of the third metatarsal bone.)

third metatarsal bones, rarely over the first or fifth. An x-ray picture, when taken immediately, frequently reveals no change but may present a fracture. Periostitis, with or without fracture, is often encountered at

an early date, but then, in all likelihood, the callus is of longer duration unless the early shadow, as Runström mentioned, is that of a subperiosteal hemorrhage. Periosteal changes are more commonly seen after ten days and fractures some weeks later. The latter, though, do not always make their appearance. Also, periosteal thickening on one or the other side of the shaft is often found associated with march foot.

Fig. 4.—Magnification to show fragmentation of the internal cuneiform bone as indicated in figure 3.

The etiology is still debatable. Periostitis and fracture are positive findings but can be considered only as the end-results of march foot.

The purpose of this paper is to describe changes in a case of march foot for which I can find no description in the literature. These changes, in addition to the usual findings, consisted of: (1) fragmentation of the internal cuneiform bone, (2) fracture of the head of a metatarsal bone and (3) fracture of the base of a metatarsal bone.

REPORT OF CASE

History.—I. A., a white man, 39 years of age, came to me complaining of pain and swelling of the left foot. On Sept. 19, 1934, ten days prior to examination, he experienced a sudden attack of pain in the arch of his left foot. Because of this pain he found it difficult to walk but continued to do so until he found it almost impossible to "carry on." For a month or so prior to this acute attack he had experienced a tiredness which was more marked toward the end of the day or when he began to walk, especially after resting. There was a history of mild infection of the left big toe about the second week of August, but this cleared up rapidly, giving him no "trouble." He gave no history of a previous attack similar to this or of any involvement of other bones or joints. The remainder of his and of the family history was not relevant.

Fig. 5.—Magnification to show fractures of the head of the second and neck of the third metatarsal bone as indicated in figure 3.

Examination.—The left foot revealed a soft tissue swelling which extended from the toes to below the ankle joint. The edema pitted only slightly to firm touch. The toes and the heads of the metatarsal bones were not sensitive to pressure. Marked tenderness was present over the fifth metatarsal bone while the remaining shaft and the other metatarsal bones were free from symptoms. There was no abnormal mobility or crepitation. Motions of the toes were not restricted or painful. Inversion of the fore part of the foot was markedly limited and resulted in pain over the base of the fifth metatarsal bone. The heel was everted and adduction of the heel almost completely limited. Flexion and extension of the ankle joint were normal. The tendons of the anterior and posterior tibial muscles were tender at the points of their insertions. The tibial and fibular

shafts were not tender. The remaining examination of both lower extremities, including the right foot, the hips, the knee and right ankle joints, gave essentially negative results. Examination of the spine and upper extremities also gave negative results.

Roentgenographic examination of the left foot (figs. 1 and 2) on September 29 showed a recent transverse fracture of the base of the fifth metatarsal bone with comminution of the proximal fragment. There was an old healed fracture in the proximal fourth of the second metatarsal shaft with some periosteal proliferation.

Fig. 6.—Right foot on Nov. 30, 1934. The medial aspect of the middle third of the third metatarsal bone presents some periosteal thickening.

A localized area of periostitis was seen on the mesial side of the middle third of the third metatarsal bone. There was some sharpness of part of the internal cuneiform bone. A small gouged out area was present on the lateral aspect of the first metatarsal head and a similar area, on the posterior articular border of the tibia. A spur projected from the inferior border of the os calcis.

The observations on the blood were as follows: hemoglobin, 14.6 Gm., or 86 per cent; red blood cells, 5,080,000; white blood cells, 7,850; polymorphonuclears,

60 per cent; small lymphocytes, 22 per cent; large lymphocytes, 4 per cent; mononuclears, 9 per cent; eosinophils, 1 per cent; bleeding time, eight and one-half minutes; coagulation time, five and one-half minutes; blood calcium, 10.4 mg.; blood phosphorus, 2.6 mg.; Wassermann and Kolmer reactions, negative.

Diagnosis.—The diagnosis was march foot.

Treatment and Clinical Course.—The foot and ankle were immobilized with adhesive strapping, and bearing of weight was not permitted. This treatment was later followed by baking and by gentle and then more vigorous massage, aiming particularly at the spasticity of the muscles. The condition improved

Fig. 7.—Left foot, anterior-posterior and lateral views, on Dec. 27, 1934. There is further evidence of fracture of the head of the second metatarsal bone with a suggestive V formation at the neck. The base of the fifth metatarsal bone and the neck of the third metatarsal bone show further evidence of healing.

gradually under treatment, and the patient began to walk with the aid of arch supports. On November 30, he complained of some discomfort in the fore part of the foot. Examination revealed distinct areas of thickening with some tenderness of the second and third metatarsal heads, immediately behind the metatarsophalangeal joints. There was no gross swelling of the toes or foot. Abduction of the heel was now only slightly limited, but inversion of the fore part of the foot was markedly limited, while motions of the toes were free. X-ray films

taken of both feet (figs. 3 to 6) revealed a healing fracture at the neck of the third metatarsal bone of the left foot with some medial displacement of the distal fragment and exuberant formation of callus. Another startling finding was the fragmentation of the internal cuneiform bone with some irregularity of part of the articular surface of the scaphoid. The distal half of the second metatarsal bone showed periosteal proliferation and a fracture of the head beginning in the lateral aspect, running transversely across immediately distal

Fig. 8.—Left foot, anterior-posterior and lateral views, on Jan. 30, 1935. There is further evidence of healing at the base of the fifth metatarsal bone, neck of the third and head of the second. The internal cuneiform bone shows greater evidence of fragmentation, and several fragments of bone can be more clearly seen on the lateral view.

to the articular surface and passing into the neck. The old fracture at the base of the fifth metatarsal bone showed healing and a faint line of periosteal proliferation. The head of the fourth metatarsal bone showed a suspicious line of fracture. The right foot showed a slight thickening on the medial aspect of the middle third of the third metatarsal shaft.

The metatarsal arch was raised, and the patient showed further improvement. Physical therapy was continued.

On December 27, examination of the left foot showed further improvement. The tenderness over the heads of the second and third metatarsal bone had disappeared; adduction of the heel was fairly free, but pronation of the fore part of the foot was still limited. An x-ray film was again taken of the left foot (fig. 7). There was evidence of further healing at the base of the fifth and neck of the third metatarsal bone. The head of the second metatarsal bone showed further evidence of a fracture, and there was also a suspicious V formation at the neck.

Fig. 9.—Right foot, anterior-posterior and lateral views, on Jan. 30, 1935. There are no changes over those found at the first examination.

The patient was reexamined on Jan. 30, 1935. He was free from pain and no longer complained of tiredness. Adduction of the heel and pronation of the fore part of the foot were free. There were no areas of tenderness. Roentgen examination of the left foot (fig. 8) showed further healing at the base of the fifth, neck of the third and head of the second metatarsal bone. The internal cuneiform bone gave greater evidence of fragmentation, and several fragments of bone could be more clearly seen on the lateral view. The x-ray film of the right foot (fig. 9) offered no changes over those observed at the previous examinations.

The changes presented by this unusual case emphasize the possibility that fragility of bone may play an important rôle in the fractures of march foot. It is not likely that similar changes had not occurred in the thousands of cases that preceded this one; in all probability some of the earlier observers overlooked them. Of still further interest is the fact that these changes partly substantiate the etiologic theories of some and deny those of others, and one may conclude that more serious thought and consideration should be given to the possibility of calcium disturbances in the bone proper.

SUMMARY

A case of march foot with several undescribed bone changes is presented. These changes were: (1) fragmentation of the internal cuneiform bone, (2) a fracture of the head of the second metatarsal bone and (3) a fracture of the base of the fifth metatarsal bone.

2309 Eutaw Place.

ACUTE APPENDICITIS AND ASSOCIATED LESIONS

SOME OBSERVATIONS ON THE MORTALITY

RUDOLPH N. SCHULLINGER, M.D.
NEW YORK

In the study of acute appendicitis for the eighteen year period between Jan. 1, 1916 and Jan. 1, 1934, at the Presbyterian Hospital in New York, it was discovered that a considerable number of the case records were classified under improper subgroups. In order to procure accurate information on the total mortality rate, as well as that of the individual subgroups, it was necessary to examine the history on every case record which had been filed under the following classifications: acute appendicitis, acute appendicitis with acute local peritonitis, acute appendicitis with peritoneal abscess, acute appendicitis with acute diffuse peritonitis and acute appendicitis with progressive fibrinopurulent peritonitis. Therefore, by careful search of these records, it was possible to present the facts which constitute this paper.

Numerous discrepancies were found between the final diagnosis listed on the record cards and the operator's description of the pathologic process or the final note. Such a discrepancy was often strengthened by the clinical symptoms and signs as noted by the house surgeon or by the attending surgeon. For instance, there were many charts on which a final diagnosis of acute appendicitis was recorded which obviously belonged to the group filed under acute appendicitis with acute local peritonitis. There were numerous case records filed under acute appendicitis with acute local peritonitis which on careful examination indicated that they should have been placed elsewhere, either under acute appendicitis or acute appendicitis with acute diffuse peritonitis or even under acute appendicitis with peritoneal abscess. This same situation obtained in the other groups studied. Sometimes two associated phases were found in one chart. This was particularly true of those filed under acute appendicitis with peritoneal abscess; in this group acute diffuse peritonitis was also recorded on seventy-seven charts; some of these were merely filed under acute diffuse peritonitis, while several were found only under the caption of peritoneal abscess, and the remainder were properly placed under both headings. Similarly, there were 2 instances of appendical abscess and progressive fibrinopurulent peritonitis which were listed only under one diagnosis. In this connection, when more than one associated lesion existed, it must be emphasized

From the First Surgical Service of the Presbyterian Hospital.

that such cases were assigned under each of the two groups to which they belonged, and if the patient died the fatality was recorded under each of these groups in the computation of the mortality rate for each separate group (chiefly in tables 1, 4, 7, 10 and 13); but in determining the total mortality rate for all the groups (table 15) these same cases in which more than one lesion was present were counted only as 1 case, and, if the patient died, only 1 death was recorded. This accounts for the fact that the sum of the individual series (tables 1, 4, 7, 10 and 13) would be greater than the total as given in table 15. Mention must also be made of those cases in which there were preoperative signs and symptoms and a gross operative pathologic process of acute appendicitis, yet in which the microscopic diagnosis failed to reveal an acute inflammatory process. The records in these cases were nevertheless filed under acute appendicitis provided the attending surgeon in his final note still considered the case as one of acute appendicitis. Several cases were found in which the history and physical signs and the appearance of the appendix did not warrant the diagnosis of an acute process, and these were excluded except in 2 or 3 instances in which the microscopic diagnosis was acute appendicitis despite the clinical evidence to the contrary.

The reader will note that acute appendicitis together with its associated lesions has been placed under five main groups (tables 1 to 14). They do not necessarily represent consecutive stages of the same disease process. Often they skip the stage of peritoneal abscess (tables 7 and 8) and proceed from one of local to one of diffuse (or spreading) peritonitis and then perhaps finally to fibrinopurulent peritonitis. There are a number of instances in which peritoneal abscess with acute diffuse peritonitis, and even with acute fibrinopurulent peritonitis, has been described at operation, although here it is likely that the abscess was present before the diffuse or fibrinopurulent peritonitis developed.

It must be realized that the five groups into which these cases have been divided are classified possibly for the purpose of convenience and study rather than as actual separate clinical entities, although in the great majority of instances the latter also probably obtains. Yet there must be many cases in which it would be extremely difficult to determine the exact category. Add to this the individual interpretation and conception of the disease process on the part of a score or more surgeons and the reader will quickly challenge the accuracy of tables 1 to 14. There is no satisfactory answer to this except to state that all questionable cases were discussed and reviewed with the attending surgeon in charge, and his decision (to which group they should belong) was considered as final. Table 15, however, represents the total number of cases of acute appendicitis of all types, together with the actual number of deaths; therefore, the figures in this table can be considered as accurate.

GROUP 1: ACUTE APPENDICITIS

Definition.—Acute appendicitis is an acute inflammatory process confined almost entirely to the appendix, in whole or in part. The organ is edematous and sometimes turgid and distended. Its serosa has lost its luster, and the serosal vessels are often dilated. There may be some deposition of fibrin and even beginning discoloration, suggesting an early stage of gangrene. The meso-appendix often presents edema, dilatation of the blood vessels and friability. The cecum, adjacent intestinal coils, mesentery, retroperitoneal tissues and parietal and visceral peritoneum show little or no inflammatory reaction. There is usually no exudate.

TABLE 1.—*Annual Mortality Rates in Cases of Simple Acute Appendicitis at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916	49	1	2.0
1917	44	0	0
1918	60	0	0
1919	43	0	0
1920	60	1 (0)*	1.66
1921	53	0	0
1922	54	1	1.85
1923	61	0	0
1924	54	1	1.85
1925	50	0	0
1926	56	0	0
1927	69	0	0
1928	74	1	1.35
1929	93	1	1.07
1930	87	0	0
1931	91	1 (0)*	1.09
1932	65	0	0
1933	112	0	0
Total	1,175 (1,173)*	7 (5)*	0.59 (0.42)*

* See analysis of deaths, page 67.

Mortality Rate.—In table 1 are listed the number of cases which conform to the definition just stated, with the number of deaths and the mortality rate. It is interesting to study the progressive five year average mortality rate for comparison, because it tends to lessen the fluctuation factor of each individual year (table 2). This is more striking in comparing the graphs in charts 1 and 2. The line in chart 2 is surprisingly level, but one notes immediately an upward progression from 0.34 per cent in the period from 1923 to 1927 to 0.73 per cent in the interval from 1928 to 1932. The latest five year average (1929-1933) has diminished markedly and is the third lowest in the series. Therefore, although the mortality rate at the Presbyterian Hospital is no higher than that of the earlier years, still there has been a slight but definite increase since 1925 up to the latest five year period. The total death rate for all the cases in this group from 1916 to 1933, inclusive, is 0.59 per cent.

Comparative Study.—Some figures from other clinics and hospitals will be interesting for comparison, and these are shown in table 3. It will be noted that the mortality rates vary considerably. Probably the most interesting figure is that of Bauer, who has made a most careful and scholarly study of cases of acute appendicitis covering a period of twenty-seven years. It did not seem fair to quote mortality rates from a personal series (such as Guerry [1]) or from a small, limited group of highly trained surgeons (Finney's [2] series, for instance), because the

TABLE 2.—*Progressive Five Year Average Mortality Rates in Cases of Simple Acute Appendicitis at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916–1920	256	2	0.78
1917–1921	260	1	0.38
1918–1922	270	2	0.74
1919–1923	271	2	0.73
1920–1924	282	3	1.06
1921–1925	272	2	0.73
1922–1926	275	2	0.72
1923–1927	290	1	0.34
1924–1928	303	2	0.66
1925–1929	342	2	0.58
1926–1930	379	2	0.52
1927–1931	414	3	0.72
1928–1932	410	3	0.73
1929–1933	448	2	0.44

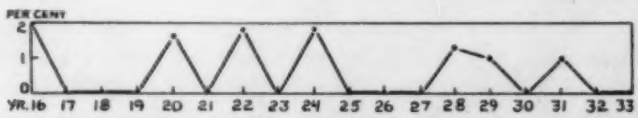


Chart 1.—Graph showing the annual death rate from 1916 to 1933, inclusive, in 1,175 cases of simple acute appendicitis. The total number of deaths was 7; the mortality rate, 0.59 per cent.

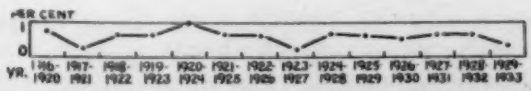


Chart 2.—Graph for comparison with that in chart 1, showing the progressive five year average death rate from 1916 to 1933, inclusive, in the same group of cases of simple acute appendicitis.

present report represents the results of a large hospital staff in which there have been at least twenty-five attending surgeons during the period under study. Many reports were found in the literature, but they are not shown in table 3 because the period of time was too short or because the series was too small. This fact also obtains in comparing the mortality

1. Guerry, Le G.: Tr. Am. S. A. **44**:136, 1926.
2. Finney, J. M. T., Jr.: Surg., Gynec. & Obst. **56**:360 (Feb.) 1933.

rates in groups 2 to 6 at the Presbyterian Hospital. Another difficult feature in endeavoring to make comparisons is that each hospital or locality has its own classification of acute appendicitis and its associated lesions. Consequently, the reader must appreciate a real factor of error in studying these charts. However, I have attempted to cite only those series which might seem to agree with the classification used in this paper. In passing, it is interesting to note that two authors (Dixon [3] and Stoney,[4] both personal series) do not have a single death in their group of cases of simple acute appendicitis.

Analysis of Deaths.—A careful study of the 7 fatal cases in this group revealed 2 in which the data furnish considerable doubt as to the validity of the cause of

TABLE 3.—*Mortality Rates in Series of Cases of Acute Appendicitis Reported in the Literature*

Year of Publication	Author	Place	Number of Cases	Period Studied	Mortality, per Cent
1925	Adams [7]	London Hospital	633	1919-1923	0.9
1925	Love [8]	St. Thomas' Hospital	348	1919-1923	0.8
1927	Colp [9]	Mount Sinai Hospital, New York	842	1916-1926	0.95
1928	Quain and Waldschmidt [10]	Bismarck, N. D.	551	1919-1927	0.36
1931	McDonald: Minnesota Med. 14: 212, 1931	St. Marys and St. Lukes Hospital, Duluth, Minn.	944	1925-1930	0.74
1933	Bauer [11]	General Hospital, Malmö, Sweden	3,750	1908-1930	0.7
1933	Walker [12]	Boston City Hospital	1,174	1927-1930	1.5
1933	Coller and McRae: J. Michigan M. Soc. 30: 319, 1931	University Hospital, Ann Arbor, Mich.	223	1925-1930	0.43
1934	Keyes: Ann. Surg. 99: 47, 1934	Barnes and St. Louis Children's Hospitals	945	1915-1932	2.32
1934	Quain: Arch. Surg. 28: 782, 1934	Bismarck, N. D.	637	Sept. 1927 to Oct. 1932	0.31

death. One patient was a 6 year old child who was admitted to the hospital with a diagnosis of intussusception. The diagnosis was verified at operation, and the appendix was found to be included in the ileocecal intussusception. After reduction was accomplished, the operator examined the appendix and thought it appeared acutely inflamed. It was therefore removed. The report from the surgical pathology laboratory was chronic appendicitis. The patient lived only ten hours after operation. It is possible that the appendix was simply congested by reason of its inclusion in the intussusception instead of being primarily acutely inflamed.

The second doubtful case was that of a 27 year old man who was admitted to the hospital because of pneumonia (type I) and a failing heart from chronic cardiac valvular disease. The abdomen was slightly distended, and no tenderness or spasm was present. The patient died five hours later. Auptosy revealed obvious pneumonia, edema of the lungs and cardiac hypertrophy as well as mitral and aortic stenosis. The abdominal cavity was clean, and the peritoneum was smooth, pale

3. Dixon, C. F.: Proc. Staff Meet., Mayo Clin. **7**:323 (June 1) 1932.
4. Stoney, R. A.: Irish J. M. Sc. **2**:645 (Nov.) 1932.

and glistening. The appendix appeared normal in its proximal two-thirds, but the distal third was slightly distended. Here the serosa was distinctly congested. Section of this portion of the appendix disclosed a thickened edematous wall, and the lumen contained a few drops of pus. The microscopic picture indicated an acute inflammatory process. One may almost certainly accept the pulmonary and cardiac pathologic processes as the primary causes of death.

Two fatalities were due to pulmonary embolism; they occurred on the fifth and on the sixth day after operation. The first fatality was that of a man, aged 29, who had a smooth postoperative course following the removal of an acutely inflamed retrocecal appendix. The other patient, an obese man of 52, suffered from marked postoperative distention. The operation lasted eighty minutes because of great difficulty in removing the appendix.

Another patient, 13 years of age, was admitted to the hospital with the diagnosis of tuberculous peritonitis and chronic ileus. A few days later rather severe pain developed in the right lower quadrant of the abdomen. Operation disclosed an acutely inflamed appendix and a complete lack of evidence of tuberculous peritonitis. Three days later there were signs of atelectasis of the left lung, together with bronchopneumonia. On the eleventh day acute ileus occurred. Cecostomy was performed because a mass, presumably inflammatory, was discovered in the transverse colon. Following this episode, a chronic cough developed with signs of bronchopneumonia over the lower lobe of the left lung. Because of the persistence of the cough and pulmonary signs, it was felt that the process might be tuberculous, but repeated examination of the sputum did not reveal any tubercle bacilli. On the fortieth day severe stomatitis set in and spread rapidly to all parts of the oral cavity, producing extensive unclean ulcers. The patient became rapidly worse and died on the fiftieth day.

The remaining 2 cases are interesting in that death might have been prevented by the use of spinal anesthesia or avertin, or even of local anesthesia. The patient in the first case was a man of 38 who had a cold and sore throat at the time of admission. Ethylene was poorly taken, so it was decided to give ether by the open method. The operation was long and difficult because of nonrotation of the cecum together with a retrocecal position of the appendix. Bronchopneumonia (type IV) developed on the fourth day. The severe coughing provoked disruption of the wound on the eighth day, and it was immediately repaired. Death followed in twenty-four hours. The other patient was a very obese man of 52 years. The operation lasted one and a half hours because of difficulty in removing a high retrocecal appendix. Ethylene-ether anesthesia was given. The postoperative course was stormy because of progressive distention and also bronchopneumonia in the lower lobe of the right lung. Jejunostomy was performed on the fourth day, at which time there was no evidence of peritonitis. The patient died twelve hours later.

It is interesting that all 7 deaths occurred in males. If one invalidates the first 2 deaths as being primarily nonappendical in origin, then the total mortality rate for simple acute appendicitis would be 0.42 per cent instead of 0.59 per cent (table 1*).

GROUP 2: ACUTE APPENDICITIS WITH ACUTE LOCAL PERITONITIS

Definition.—The condition is an acute inflammatory process which is not confined to the appendix alone but involves the viscera in the close vicinity as well as the adjacent parietal and visceral peritoneum. In

addition to the pathologic process noted in the previous group, there may be considerable deposition of fibrin on the appendix. The latter may show more advanced signs of gangrene. There is usually no gross perforation. The cecum about the base of the appendix may be edematous, thickened and congested. The omentum, which is edematous and congested, is sometimes found partly or wholly enveloping the appendix, perhaps with the formation of a few delicate adhesions. The adjacent intestinal coils may be slightly dilated, and sometimes there are congestion and loss of luster of the serosal surface. This same appearance may be found on the mesentery of the terminal portion of the ileum.

TABLE 4.—*Annual Mortality Rates in Cases of Acute Appendicitis with Acute Local Peritonitis at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916	29	0	0
1917	29	0	0
1918	25	2	8.0
1919	29	0	0
1920	30	1	3.33
1921	41	2 (1)*	4.87
1922	35	1	2.85
1923	35	1	2.85
1924	32	1	3.12
1925	34	0	0
1926	45	2 (1)*	4.44
1927	33	0	0
1928	34	1	2.94
1929	40	0	0
1930	20	1	5.0
1931	22	0	0
1932	77	0	0
1933	40	0	0
Total	630 (628)*	12 (10)*	1.90 (1.59)*

* See analysis of deaths, page 72.

Some peritoneal exudate is almost always present. It may be confined to the right lower quadrant or perhaps only to the iliac fossa; on the other hand, there may be a small collection in the pelvis in addition. This exudate may be clear, pale and odorless, but it is often turbid and grayish yellow and possibly slightly odorous. Sometimes it appears as a brownish, seropurulent fluid, but it is rarely bloody, frankly purulent or foul-smelling. The parietal peritoneum overlying the involved area may be edematous and injected. Briefly, the process is more extensive than that of the previous group, yet it tends to remain relatively confined to a limited area of the peritoneal cavity.

Mortality Rate.—Table 4, together with charts 3 and 4, gives the facts concerning the annual death rate and the five year average. Here again, chart 3 shows a fluctuating curve. In chart 4 one is

impressed by a definite, general decline, which reaches its lowest point in the last five year period. There are a few rises interspersed along the curve, the most serious being during the period from 1918 to 1922, inclusive. The mortality rate in the group of acute appendicitis with acute local peritonitis is definitely decreasing up to the present time; perhaps it will continue to do so. The total death rate in this group is 1.9 per cent, but the rate for the most recent five year period is only 0.5 per cent.

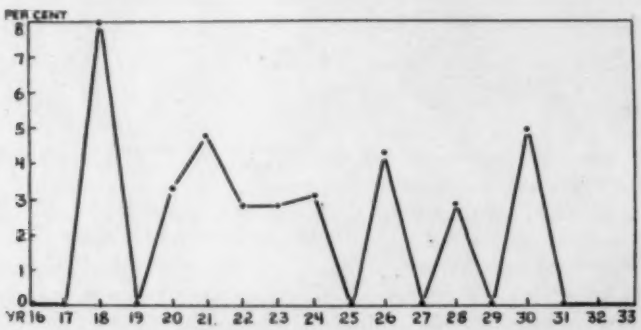


Chart 3.—Graph showing the annual death rate from 1916 to 1933, inclusive, in 630 cases of acute appendicitis with acute local peritonitis. The total number of deaths was 12; the mortality rate, 1.9 per cent.

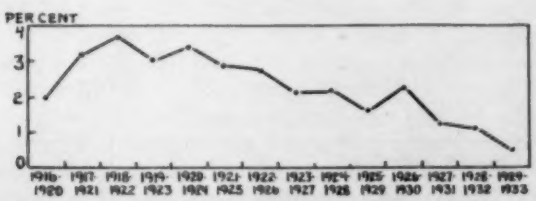


Chart 4.—Graph for comparison with that in chart 3 showing the progressive five year average death rate from 1916 to 1933, inclusive, in the same group of cases of acute appendicitis with acute local peritonitis.

Comparative Study.—Examination of mortality reports from other clinics indicates that the death rate at the Presbyterian Hospital compares rather favorably with them. This is shown in table 6. Even the mortality rates in the personal series of Ashhurst [5] (4.5 per cent), Stoney [4] (2.2 per cent) and Hawe [6] (2.8 per cent) are slightly higher.

Analysis of Deaths.—There were 12 deaths in this group of cases. In 2 the cause of death seems doubtful from the standpoint of acute appendicitis with acute

5. Ashhurst, A. P. C.: Ann. Surg. **85**:89 (Jan.) 1927.
6. Hawe, P.: Liverpool Med.-Chir. J. (pt. 1) **41**:89, 1933.

local peritonitis. The first case was that of a 9 year old boy who was observed for four days after admission to the hospital because of extremely confusing signs, which seemed to point to meningitis. Abdominal examination failed to reveal any signs of acute peritoneal irritation. The boy was extremely irritable; his neck was slightly stiff; there was a questionable Kernig sign, but examination of the fundi and spinal fluid gave negative results. The white blood cells numbered 31,000, with 84 per cent polymorphonuclears, and a culture of the blood showed a growth of the hemolytic streptococcus. The temperature remained around 104 F. Finally, on the fourth day, signs of tenderness in the lower portion of the abdomen and slight involuntary muscle spasm developed. At operation the picture was one of acute mesenteric adenitis; there was a small amount of clear, odorless, sticky exudate. The appendix was retrocecal and did not appear to be inflamed. However, the surgical pathology laboratory reported a diagnosis of acute appendicitis. The patient died two days later, having lapsed into coma and acidosis. Permission for autopsy was not obtained.

The second doubtful case was that of a girl, aged 14 years, who was admitted to the hospital with a diagnosis of acute appendicitis. There was an elevated blood count and an increase in the number of polymorphonuclears. At operation, acute local peritonitis was discovered. The appendix was laterocecal, and the distal two thirds was gangrenous; it was decided to use a drain. The postoperative course was uneventful, and the patient was discharged on the fifteenth day with instructions to visit the surgical clinic because the wound had not quite healed. During the following five weeks the wound showed no tendency to heal; in fact, it became slightly deeper. It had an unclean appearance, with unhealthy, grayish granulations and a thin, seropurulent discharge. During the fifth week the girl felt weak and feverish and lost her appetite. When the next dressing was applied, her general appearance was found to be distinctly poor; the temperature was 100 F. and, to the astonishment of the examiner, the white blood count was 15,000, with 88 per cent large lymphocytes and immature forms. Further investigation showed an enlarged spleen and liver, but there was little or no general adenopathy. The patient was readmitted to the hospital and observed for two days, and then, because of increasing pain in the right lower quadrant, together with rising temperature and an increasing white cell count, the wound was explored, but no foreign body or residual abscess was found. A culture of the wound tract showed a growth of hemolytic streptococcus. The patient became rapidly worse; a blood culture on the third day after operation was positive for the hemolytic streptococcus. Subsequent blood counts revealed rapidly developing aleukemic leukemia. Petechiae soon appeared, and just before death there was severe ulcerative stomatitis. The final report of the autopsy and microscopic examination was acute lymphatic leukemia and hemolytic streptococcic bacteremia. It was the pathologist's belief that the leukemia probably existed before the episode of acute appendicitis took place.

If the cause of death in these 2 cases can be assigned to sources other than acute appendicitis with acute local peritonitis, the total mortality rate would be 1.59 per cent (table 4*).

The remaining 10 deaths deserve some discussion. Six of the patients were aged 20 years or less. All 10 gave a relatively short history (with 1 exception). A recent acute infection of the respiratory tract had occurred in 2, and 1 patient gave a history of several attacks of pneumonia. As 1 man was an extensive user of alcohol, it was necessary to administer ether by the open method. Four patients received nitrous oxide and ether, and another was given chloroform and ether. Ethylene was used once. The anesthesia in the remaining case was started with

nitrous oxide and ether but, because of cyanosis and spasm, ether by the open method was substituted. The position of the appendix varied considerably. More significant are the following facts: Removal of the appendix was extremely difficult and the operation was considerably prolonged in 3 cases (either by reason of the obesity of the patient, poor relaxation and response to the anesthetic, or because of the position of the appendix itself involving serious technical difficulties); the large bowel was accidentally opened in 1 case, and in 2 the appendix ruptured during the process of removing it. These are all serious accidents, and they furnish thought for means of prevention. Severe distention and ileus (mechanical in 2) developed in 7 cases, necessitating enterostomy or jejunostomy. This operation was usually done as a last desperate and final effort. It seems that jejunostomy might produce a more fortunate outcome if done earlier, as soon as other more conservative measures fail. This is one reason that enterostomy is condemned by many surgeons. One patient died of pylephlebitis, and in another the same complication developed together with a hepatic and subphrenic abscess. A secondary peritoneal abscess occurred in 2 cases. Urinary suppression was the cause of death in another case. Postoperative pneumonitis was a serious complication in 4 instances and undoubtedly the cause of death in 1. Drainage was established in all but 1 of the 10 cases at the original operation.

TABLE 5.—*Progressive Five Year Average Mortality Rates in Cases of Acute Appendicitis with Acute Local Peritonitis at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916–1920	142	3	2.11
1917–1921	154	5	3.24
1918–1922	160	6	3.75
1919–1923	170	5	2.94
1920–1924	173	6	3.46
1921–1925	177	5	2.82
1922–1926	181	5	2.76
1923–1927	179	4	2.23
1924–1928	178	4	2.24
1925–1929	186	3	1.60
1926–1930	172	4	2.32
1927–1931	149	2	1.34
1928–1932	193	2	1.03
1929–1933	199	1	0.50

TABLE 6.—*Mortality Rates in Series of Cases of Acute Appendicitis with Local Peritonitis Reported in the Literature*

Year of Publication	Author	Place	Number of Cases	Period Studied	Mortality, per Cent
1925	Adams [7]	London Hospital	467	1919-1923	6.2
1925	Love [8]	St. Thomas' Hospital	271	1919-1923	5.2
1927	Colp [9]	Mount Sinai Hospital, New York	975	1916-1926	3.1
1933	Walker [15]	Boston City Hospital	265	1927-1930	1.5
1933	Seelye [18]	Memorial Hospital, Worcester, Mass.	287	1929-1932	1.4
1934	Bower: J. A. M. A. **102**: 813 (March 17) 1934	Philadelphia hospitals	3,817	1928-1932	2.50
1934	Garlock: Am. J. Surg. **23**: 248, 1934	New York Hospital II Division	350	1928-July 1932	0.57

It seems that certain preventive measures might be instituted for lowering the mortality rate in this particular group. Great care should be given to the choice of an anesthetic, especially in the presence of an infection of the upper respiratory tract, emphysema, obesity and alcoholism. At the present time the use of spinal anesthesia, or avertin with nitrous oxide or local anesthesia, seems highly desirable. Prevention of injury to the neighboring viscera and gentle removal of the appendix in order to avoid rupturing it are technical points which require no further comment. When there is actual difficulty in removing the appendix, it might be wiser simply to insert a drain down to it, because if removal is attempted there may be considerable damage to the stump of the meso-appendix and the retroperitoneal tissues, thereby affording a means of extension of the infection and producing, possibly, pylephlebitis, retroperitoneal cellulitis, phlebitis of the retroperitoneal veins and even septicemia. This certainly suggests itself as a possible cause in the 2 cases in which pylephlebitis and hepatic abscess occurred. If enterostomy is to be employed, it should be done early and not as an eleventh hour desperate measure. Lastly, it is interesting to note that a number of patients with streptococcic appendicitis and local peritonitis give a history on admission of high fever, diarrhea and chill. These are the persons in whom a severe, overwhelming infection is likely to develop, with septicemia, pylephlebitis, subsequent general peritonitis or paralytic ileus. They are the ones who need large amounts of fluids to combat the toxemia, and, equally important, they should receive repeated small blood transfusions (300 cc.) as a part of the supportive treatment.

If these few simple preventive measures are employed in this group of cases of appendicitis, it seems reasonable to suppose that the mortality rate will drop considerably more.

GROUP 3: ACUTE APPENDICITIS WITH PERITONEAL ABSCESS

Definition.—In cases of acute appendicitis with peritoneal abscess the process has remained localized, as a rule, because of resistant forces which have prevented further spread of the infection. The abscess may be formed by any or all adjacent viscera, and its location depends, in large part, on the position of the appendix. The latter is usually found, in whole or in part, in the abscess cavity. It frequently reveals one or more perforations, and its surface may be covered with fibrin. There is often extensive gangrene, and sometimes the appendix may have partly or completely sloughed away. The contents of the abscess consist of thick, creamy, purulent exudate, containing fibrin and sometimes gas bubbles. The pus is often very odorous, and its color is yellowish, as a rule, although it may assume a greenish or even a brownish hue. Sometimes the abscess is very small and may contain 1 cc. of pus or less; more often one encounters several drachms or even several ounces of

pus. The very large abscesses are seen (fortunately) with less frequency, but they sometimes acquire an enormous size, even larger than a baby's head. In abscesses of recent formation the walls are apt to be made up of friable, edematous, easily bleeding tissue, and there is relatively little fibrin. The adhesions between neighboring viscera, forming the wall of the abscess, can be separated with alarming ease. In the larger and older abscesses the wall is often thicker, owing to considerable deposition of fibrin; frequently the fibrin gives a shaggy appearance to the inner surface of the wall of the abscess. Here the adhesions of the surrounding structures are stronger, and if the operator (often

TABLE 7.—*Annual Mortality Rate in Cases of Acute Appendicitis with Peritoneal Abscess at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916	39	3	7.69
1917	40	1	2.50
1918	38	2	5.26
1919	44	7 (4)*	15.90
1920	43	9 (4)*	20.93
1921	37	3	8.10
1922	29	6 (2)*	20.68
1923	52	2	3.84
1924	42	6 (1)* (1)†	14.28
1925	27	2	7.40
1926	25	2 (2)*	8.0
1927	17	1	5.88
1928	12	0	0
1929	15	2 (2)*	13.33
1930	27	4 (2)* (1)†	14.81
1931	23	3 (2)*	13.04
1932	32	5 (2)*	15.62
1933	29	2 (1)*	6.89
Total	571 (492)* (490)†	60 (38)* (36)†	10.5 (7.72)* (7.34)†

* See analysis of deaths, page 79.
† See analysis of deaths, page 79.

inadvisedly) digitally tears the wall of the abscess, there is less danger of bleeding and of spreading the infection. One portion of the abscess is almost always formed by the parietal peritoneum, so that there may be considerable retroperitoneal cellulitis with thrombosis of the veins. And this same condition often obtains for the thickened, edematous, friable, thrombosed meso-appendix. Around the abscess there may be edema and congestion of the viscera, and one usually notes a small amount of free serous or slightly turbid peritoneal exudate. If there has been extension of the infection through the wall of the abscess, there may be an associated diffuse (spreading) or even general fibrinopurulent peritonitis.

Mortality Rate.—Examination of table 7 and also of charts 5 and 6 shows a definite disturbing increase in the mortality rate in recent years. Chart 6 illustrates this quite obviously, proceeding from a low level of

7.29 per cent in the period from 1925 to 1929 steadily upward to 12.69 per cent in the last five year period. While the average death rate in this group of cases is 10.5 per cent, there is reason to view this rise with alarm. On the other hand, the annual mortality rate discloses the lowest death rate in 1933 since 1928, and, if this can be maintained, the curve should begin to drop sharply.

Comparative Study.—Table 9 discloses data from other hospitals and clinics for comparison with those from the Presbyterian Hospital. The

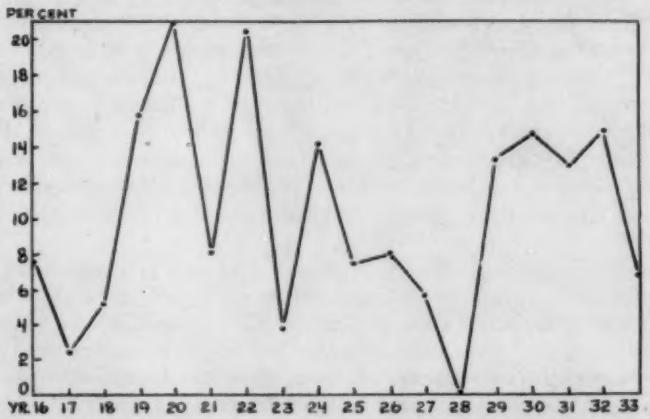


Chart 5.—Graph showing the annual death rate from 1916 to 1933, inclusive, in 571 cases of acute appendicitis with peritoneal abscess. The total number of deaths was 60; the mortality rate, 10.5 per cent.

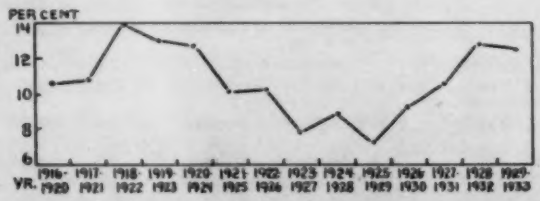


Chart 6.—Graph for comparison with that in chart 5, showing the progressive five year death rate from 1916 to 1933, inclusive, in the same group of cases of acute appendicitis with peritoneal abscess.

mortality rate from the latter hospital is considerably higher than that given by Adams,[7] Love,[8] Colp,[9] Quain and Waldschmidt [10] and Bauer.[11]

7. Adams, J. E.: Brit. M. J. **1**:723 (April 18) 1925.

8. Love, R. J.: Lancet **1**:1229 (June 10) 1933.

9. Colp, R.: Ann. Surg. **85**:257 (Feb.) 1927.

10. Quain, E. P., and Waldschmidt, R. H.: Acute Appendicitis: Report of 1,000 Consecutive Cases, Arch. Surg. **16**:868 (April) 1928.

11. Bauer, G.: Acta chir. Scandinav. (supp. 24) **70**:1, 1933.

Of particular interest is the report of Sworn and Fitzgibbon,[12] in which the death rate in a series of cases in which operation had been performed is compared with that in a series in which there was no operation. The nearest approach to this is the personal series of Guerry,[1] with a mortality of 0.7 per cent. Seelye,[13] in a small group of cases covering a

TABLE 8.—*Progressive Five Year Average Mortality Rates in Cases of Acute Appendicitis with Peritoneal Abscess at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916-1920	204	22	10.78
1917-1921	202	22	10.89
1918-1922	191	27	14.13
1919-1923	205	27	13.17
1920-1924	203	26	12.80
1921-1925	187	19	10.16
1922-1926	175	18	10.28
1923-1927	163	13	7.97
1924-1928	123	11	8.94
1925-1929	96	7	7.29
1926-1930	96	9	9.37
1927-1931	94	10	10.63
1928-1932	109	14	12.84
1929-1933	126	16	12.69

TABLE 9.—*Mortality Rate in Series of Cases of Acute Appendicitis with Abscess Reported in the Literature*

Year of Publication	Author	Place	Number of Cases	Period Studied	Mortality, per Cent
1925	Adams [7]	London Hospital	347	1919-1923	4.6
1925	Love [8]	St. Thomas' Hospital	95	1919-1923	4.2
1927	Colp [9]	Mount Sinai Hospital, New York	665	1916-1926	5.4
1928	Quain and Waldschmidt [10]	Bismarck, N. D.	289	1919-1927	2.42
1931	Tasche and Spano: Ann. Surg. 93: 899, 1931	University Hospital, Minneapolis	112	1920-1929	9.7
1932	Sworn and Fitzgibbon [12]	St. Thomas' Hospital No operation	189 285	1920-1929 1920-1929	7.9 0.68
1933	Walker [15]	Boston City Hospital	328	1927-1930	9.7
1933	Bauer [11]	General Hospital Malmö, Sweden	676	1908-1930	6.9
1934	Keyes: Ann. Surg. 99: 47, 1934	Barnes and St. Louis Children's hospitals	315	1915-1932	7.3
1934	Garlock: Am. J. Surg. 23: 248, 1934	New York Hospital, II Division	51	1928-July 1932	11.76

period of only three years, reported no deaths. The remaining observers, listed in table 9, reported a mortality comparable to that from the Presbyterian Hospital.

Analysis of Deaths.—Of a total of 571 patients, 60 died. However, there were 77 patients who had acute diffuse peritonitis at operation, in addition to abscess,

12. Sworn, B. R., and Fitzgibbon, G. M.: Brit. J. Surg. **19**:410 (Jan.) 1932.
13. Seelye, W. C.: New England J. Med. **208**:123 (Jan. 19) 1933.

and of these 20 died. Similarly, there were 2 patients with acute progressive fibrinopurulent peritonitis, together with abscess, and both died. On examining the records for these 22 fatal cases, there was no question but that death was due to the more serious diffuse, or fibrinopurulent, peritonitis, and not primarily to the abscess. These cases are listed under groups 4 and 5, where they properly belong, but they have also been placed in the present series in order to study appendical abscess as a whole and to avoid the criticism of deliberately trying to reduce the mortality rate for appendical abscess. For if these 79 records and the 22 deaths associated with them are excluded—and many might consider this as fair and justifiable—the mortality rate would be 7.72 per cent instead of 10.5 per cent (table 7*). In addition to these 79 cases, there are 2 more which are certainly questionable when the cause of death is listed as appendical abscess. The first case was that of a woman aged 68, who was admitted to the hospital with a palpable appendical abscess. She had had a stroke three years before. The abscess was drained under infiltration with procaine hydrochloride, and the postoperative course was smooth until the occurrence of a cerebral accident on the tenth day. The patient became disoriented, confused and incontinent. Facial paralysis on the right side appeared on the sixteenth day, and she died of terminal bronchopneumonia three days later. Death can hardly be directly attributed to the peritoneal abscess.

The second case was that of a man aged 34, who was extremely ill and in a poor condition. An operation for an appendical abscess was performed at another hospital three months before. The patient was well for about a month and then began to have increasing fever and chills, together with intermittently draining sinuses. He was almost moribund when he entered the Presbyterian Hospital, and in spite of supportive treatment he died two days later. Autopsy revealed a subcecal abscess, thrombophlebitis of the pelvic veins, pylephlebitis, abscess of the liver and bronchopneumonia. As this patient was operated on elsewhere and came to the hospital too ill for any further surgical intervention, it does not seem fair to include his case in computing the mortality rate of this group. If these 2 cases are excluded, as well as the 79 mentioned previously with 22 deaths, then the total number in this series would be 490 with 36 deaths, carrying a mortality of 7.34 per cent (table 7†). Nevertheless, these 24 fatal cases have been included in computing the following facts in the present death analysis:

Thirty-six of the 60 patients who died were males. Forty years was the average age, and only 18 were 30 years old or less. The youngest patient was 5½ years of age. The great majority of cases occurred in the fourth, fifth and sixth decades. The average duration of the present illness was six days. There was a recent infection of the respiratory tract in 9 cases, and active pulmonary tuberculosis in 2. Diabetes was present in 2 other cases, and marked obesity was observed in 10. A mass was felt in 25, and in 4 others it was detected on rectal examination only. During the first few years ether (by the open or closed method) was used; later, nitrous oxide, with or without ether, was used, and then ethylene, sometimes with ether. In more recent years, avertin or spinal anesthesia has been employed. Local infiltration with procaine hydrochloride has been employed only occasionally. The four most frequent sites of the peritoneal abscess were latero-cecal, subcecal and retrocecal, mesocecal and pelvic. The abscess was described as "large" in 16 and "small" or "moderate-sized" in the remaining 44 patients. The appendix was removed in 45 of the 60 patients; removal was sometimes extremely difficult, thereby injuring natural barriers against the spread of the infection, often producing bleeding and lengthening the operating time considerably. The complications were chiefly intraperitoneal, pulmonary and vascular. An over-

whelming infection, with spreading peritonitis and severe toxemia, was described in 17. Distention was marked in 26 patients, and of these 14 had a paralytic ileus. Mechanical ileus developed in 5, and there was gastric dilatation in 6 others. Enterostomy was performed four times during the original operation and eleven times as a subsequent procedure. Secondary peritoneal abscess, usually late, occurred in 7 cases, and abscess of the liver was noted at autopsy in another 3. Of the 5 patients with pylephlebitis, 4 exhibited jaundice. Severe hemorrhage and disruption of the wound each occurred in 3 cases. Sixteen patients contracted postoperative pneumonitis, while 3 died of pulmonary tuberculosis. Four died of sudden fatal embolism. Severe profound shock was noted in 5. Pericarditis, suppurative parotitis and apoplexy were each present once. Two patients died of acidosis with diabetic coma, and 4 others of uremia. The great majority of these patients died within the first week or ten days.

A few cases deserve special mention. One patient underwent ileocolectomy because the inflammation, induration and thickening of the cecum and terminal portion of the ileum were interpreted as tuberculosis. This could not be substantiated by the surgical pathologist. Another patient suddenly went into a terminal toxic psychosis and died about a week later. A very interesting case was that of a patient admitted to the hospital with a diagnosis of pneumonia. The abdominal symptoms were completely masked by the pulmonary condition. The course was progressively downhill, with the development of marked distention. Roentgenograms failed to verify the clinical diagnosis of pneumonia. Finally, signs of subphrenic abscess appeared, and a transpleural thoracotomy was performed on the fifteenth day after admission. Autopsy on the following day revealed acute appendicitis with a pelvic abscess, abscesses of the liver and pylephlebitis. Another patient probably died from the premature injection of surgical solution of chlorinated soda (Dakin's solution) into the abscess cavity. A spreading peritonitis followed, and the patient died on the sixth day. A man, aged 63, came to the surgical service with a vague history of abdominal pain. A firm, rather tender mass was felt in the ileocecal region, and it was thought that it represented a carcinoma of the cecum. The patient was observed for eleven days. At operation a large retrocecal and laterocecal abscess was entered, obviously of appendical origin. The appendix was removed. Postoperative pneumonia soon appeared; jaundice developed on the fifth day, and the patient died the following morning. The record of a 17 year old boy is noteworthy. There was a history of illness of four days' duration which, combined with the physical examination, suggested a subsiding process. Because of an existing acute tonsillitis, it was considered safe to wait. On the tenth day sudden pain and cramps developed over the lower portion of the abdomen. Operation was immediately performed. A subcecal abscess was found. Appendectomy was very difficult. Marked distention and signs of spreading peritonitis set in the following day, to be followed by hiccup, vomiting and gastric dilatation. The patient died on the second day after operation. Another somewhat similar case was that of a woman, aged 79, with a history of abdominal pain of seven days' duration. She was very obese, and abdominal examination was extremely difficult to interpret. Operation followed three days later. The anesthetic was extremely poorly taken. The operator found a large retrocolic abscess, communicating with a mesocolic abscess. The appendix was removed. There was considerable retroperitoneal cellulitis in the vicinity of the abscesses. The patient died twelve hours later. Finally, one must not neglect to mention the case of a man who was operated on for acute appendicitis and drainage of a small pelvic abscess. Six days later signs of an acute mechanical ileus suddenly developed, and after all conservative measures failed to relieve him, he was taken to

the operating room to undergo jejunostomy. During the operation, while under anesthesia, the patient vomited and aspirated a considerable portion of the contents of the stomach. He died a few hours later from a fulminating aspiration pneumonia.

This analysis of the deaths and description of some of the cases constrains one to suggest certain preventive measures in trying to reduce the mortality rate. An older age group is being dealt with, which increases the hazard of operation and anesthesia. Would it not be wiser, therefore, to use local anesthesia oftener, especially in those cases in which a mass is palpable and in which it seems close to the anterior abdominal wall? Here, too, spinal anesthesia or avertin seems preferable to general anesthesia. In the operative procedure itself one should aim to create the least possible trauma and do the simplest and quickest sort of operation. Therefore, why not simply drain the abscess? Removal of the appendix under these circumstances is often difficult; it prolongs the operation and is fraught with dangers: It may provoke the inception of a retroperitoneal cellulitis, and often the trauma of appendectomy results in breaking through protective barriers. If the incision of choice is a right rectus incision, it is well to insert a few supporting stay sutures when sewing up the wound, because the distention, coughing, vomiting, straining and gastric lavage which often follow soon after operation may provoke disruption of the wound. By reason of the severe infection in these cases, it is probably better judgment not to approximate the subcutaneous tissues and the skin; these wounds should not be sewn too tightly. The surgeon must avoid the early irrigation of the abscess and wound with surgical solution of chlorinated soda, because there is great danger of producing a sudden spread of the infection into the general peritoneal cavity. One should wait at least five days after operation before instituting this procedure, and if its introduction causes severe abdominal pains it should be stopped at once. Because of the severe toxemia and infection, it is important that these patients receive large amounts of fluid by clysis, infusion, rectal tap and perhaps continuous intravenous drip. The fluid used should be chiefly a saline solution. One or more small transfusions are extremely helpful in combating an overwhelming infection and shock; one should not wait for complications to develop; one must learn to anticipate them and then direct one's efforts and therapy against their occurrence. If enterostomy or jejunostomy is planned, it should be done early and not after distention and paralytic ileus have existed for several days. One case of disruption illustrates the advisability of enlisting the support of a snug abdominal binder before performing a gastric lavage. In order to avoid aspiration pneumonia, it is extremely important to insist on a preoperative lavage in cases of ileus. Further, the 3 cases which were under observation before operation (described in the

analysis of deaths) emphasize certain definite risks and dangers in temporizing. Sometimes there is no other choice; the surgeon must learn to realize the gravity and seriousness of the situation and be prepared to intervene at once if the patient shows signs of spreading infection. Lastly, it is of the greatest importance to group the treatments with rest between times.

In this group also there was a relatively high incidence of pulmonary embolism and pylephlebitis; this can be easily understood if the pathologic process of appendical abscess, as described in the preceding pages, is accepted. Once more, therefore, let it be urged that the utmost gentleness be exercised in the operative procedure and an effort be made to avoid opening new avenues and routes to bacterial invasion. One must try to preserve the protective barriers already set up by the patient's resisting forces. One should help, not antagonize, nature's protective mechanism.

GROUP 4: ACUTE APPENDICITIS WITH ACUTE DIFFUSE (DIFFUSING, SPREADING) PERITONITIS

Definition.—In this group of cases the process has spread from a state of local peritonitis, or of abscess formation, to one of extensive involvement of the abdominal cavity. The parietal and visceral peritoneum are usually edematous, markedly congested and somewhat friable throughout the operative field; perhaps all, or the greater part, of the celom is thus affected. The serosa of the abdominal viscera has lost its luster, and there may be moderate dilatation of the intestinal coils. Considerable purulent exudate is seen in all or most of the quadrants; it is usually thin and pale, but sometimes it is thick and creamy or reddish brown. The pus may be odorless, but frequently it is foul-smelling. As yet, there is little or no deposition of fibrin, so that few or no adhesions are seen, although there may be considerable fibrin, with adhesions, in the vicinity of the appendix itself, especially if the diffuse peritonitis is preceded by the stage of abscess formation. The appendix may be partly or completely gangrenous and covered with shreds or plaques of fibrin. In the majority of cases, one or more perforations are seen in the appendix, and sometimes a fecolith may be discovered there. Not infrequently these fecoliths may be found in the abscess or in the peritoneal cavity. The meso-appendix is usually congested, thickened, edematous and very friable, and its blood vessels are often thrombosed. Retroperitoneal cellulitis may have developed around it. That portion of the cecum surrounding the base of the appendix may be indurated, edematous and congested, so that slight trauma may provoke tearing and bleeding of its serosa. The omentum also exhibits an extensive inflammatory reaction, and it may have formed adhesions in the region of the appendix in its effort to localize

the infection. In short, the process has become a rapidly spreading or diffuse one, and pus is seen not only in the area about the appendix and pelvic cavity but also in the other quadrants, in the lumbar gutters and, occasionally, even in the subhepatic and subphrenic spaces.

Such an intra-abdominal pathologic process usually produces a fairly constant clinical picture. The patient looks acutely ill and often toxic, and the skin is hot and dry. The temperature is distinctly elevated and is between 101 and 103 F. The face is often flushed, and there may be slight dyspnea. Abdominal examination discloses a marked decrease, or an absence, of respiratory excursions. There are generalized involuntary muscle spasm, tenderness and rebound tenderness, and these

TABLE 10.—*Annual Mortality Rates in Cases of Acute Appendicitis with Acute Diffuse Peritonitis at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916	13	5	38.46
1917	17	3	17.64
1918	13	0	0
1919	20	6	30.0
1920	31	6	19.35
1921	19	1	5.26
1922	28	8	28.57
1923	20	2	10.0
1924	20	3	15.0
1925	16	0	0
1926	20	4	20.0
1927	14	2	14.28
1928	12	2	16.66
1929	14	3	21.42
1930	23	5	21.73
1931	15	2	13.33
1932	22	3	13.63
1933	12	1	8.33
Total	329	56	Average: 17.02

signs may exhibit their maximum intensity in the right lower quadrant, suprapubic region or umbilical or periumbilical area, or more rarely in the right upper quadrant or left lower quadrant. This very fact alone, that the area of maximum intensity is not always found in the right lower quadrant, makes the early diagnosis in some of these cases extremely difficult. The appendix is often of the midline or pelvic type, thus favoring an easier and more rapid spread of the infection, so that there may be a complete lack of any localizing signs. It often requires great skill on the part of an experienced surgeon, let alone the general practitioner, to diagnose this condition early in its course.

Mortality Rate.—The mortality rate is distinctly higher than that of the previous group (abscess), and a study of table 10 and charts 7 and 8 illustrates this clearly. Although the annual death rate percentages and the curve in chart 7 show a recent diminution in the

mortality, the progressive five year averages (table 11 and chart 8) are disturbing in that there is only a slight decline in the last three five year periods, and they do not compare favorably with some of the earlier ones. In fact, it is evident on studying the progressive five year average in table 11 and chart 8 that the mortality from acute appendicitis with

TABLE 11.—*Progressive Five Year Average Mortality Rates in Cases of Acute Appendicitis with Acute Diffuse Peritonitis at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916–1920	94	20	21.28
1917–1921	100	16	16.0
1918–1922	111	21	18.91
1919–1923	118	23	19.49
1920–1924	118	20	16.94
1921–1925	103	14	13.59
1922–1926	104	17	16.34
1923–1927	90	11	12.22
1924–1928	82	11	13.41
1925–1929	76	11	14.47
1926–1930	83	16	19.27
1927–1931	78	14	17.94
1928–1932	86	15	17.44
1929–1933	86	14	16.27

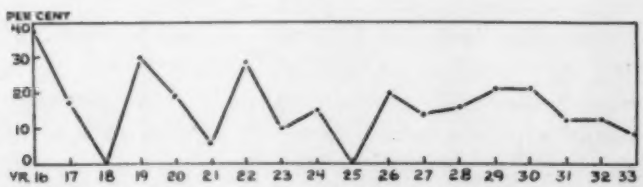


Chart 7.—Graph showing the annual death rate from 1916 to 1933, inclusive, in 329 cases of acute appendicitis with acute diffuse peritonitis. The total number of deaths in this group was 56; the mortality rate, 17.02 per cent.

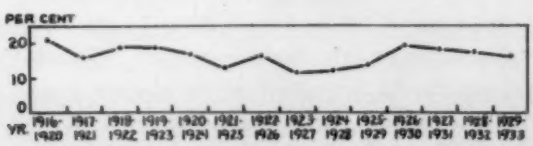


Chart 8.—Graph for comparison with that in chart 7, showing the progressive five year average death rate in the same group of cases of acute appendicitis with acute diffuse peritonitis.

acute diffuse peritonitis has definitely increased when compared to the earlier five year periods between 1921 and 1929. The reader will note that the total average mortality rate of 17.02 per cent (table 10) is almost 1 per cent higher than that of the most recent five year period (16.27 per cent).

Comparative Study.—The statistics for this group from the Presbyterian Hospital compare favorably with those of other investigators (table 12). Potter [14] reported a mortality of 35 per cent in 63 cases of acute appendicitis with acute diffuse peritonitis at the Bellevue Hospital, first surgical division, from 1920 to 1931. However, in the thirty-one months prior to the report of his most recent paper there was a mortality of only 15 per cent, which is about the same as that at the Presbyterian Hospital for that same period (12 per cent). Among those reporting a relatively large series of cases, only Bauer [11] and Walker [15] reported mortality rates which are lower than that at the Presbyterian Hospital. Many of the personal series, as well as those reported by a small limited group of surgeons, showed percentages as high or

TABLE 12.—*Mortality Rates in Series of Cases of Acute Appendicitis with Acute Diffuse Peritonitis Reported in the Literature*

Year of Publication	Author	Place	Number of Cases	Period Studied	Mortality, per Cent
1925	Adams [7]	London Hospital	230	1919-1923	20.0
1925	Love [8]	St. Thomas' Hospital	187	1919-1923	29.4
1927	Colp [9]	Mount Sinai Hospital New York	350	1916-1926	20.0
1928	Quain and Waldschmidt [10]	Bismarck, N. D.	160	1919-1927	10.0
1932	Sworn and Fitzgibbon [12]	St. Thomas' Hospital	231	1920-1929	20.0
1933	Bauer [11]	General Hospital, Malmö, Sweden	569	1909-1930	10.7
1933	Walker [15]	Boston City Hospital	204	1927-1930	11.8
1934	Bower: J. A. M. A. **102**: 813 (March 17) 1934	Philadelphia Hospitals	2,118	1928-1932	27.47
1934	Potter [14a]	Bellevue Hospital, I Division, New York	63	1920-1931	35.0
1934	Potter [14b]	Bellevue Hospital, I Division, New York	20	July 1931 to Feb. 1934	15.0

higher (Finney,[2] 22.08 per cent; Ashhurst,[5] 16.1 per cent, and Black,[16] 18 per cent). On the other hand, there are other personal series which are considerably lower (Dixon,[3] 10.4 per cent; Guerry,[1] 8.2 per cent [1.6 per cent for the group in which operation was not performed], and Stoney,[4] 8.1 per cent).

Analysis of Deaths.—The analysis of deaths and comments on possible means of mortality reduction in this very interesting and important group will be presented in detail in another paper. However, one may state that the outstanding measures of lowering the death rate in this particular group are adequate preoperative preparation, proper choice of anesthesia, the simplest operative procedure with minimum trauma,

14. Potter, P. C.: (*a*) S. Clin. North America **14**:379 (April) 1934; (*b*) Ann. Surg. **99**:985 (June) 1934.

15. Walker, I. J.: New England J. Med. **208**:113 (Jan. 19) 1933.

16. Black, J. M.: Brit. J. M. **2**:1136 (Dec. 24) 1932.

speed (but not at the sacrifice of care and precision) and thoughtfully planned postoperative measures directed particularly against shock, distention and paralytic ileus and toxemia from an overwhelming infection. The recent papers by Jones, advocating appendicostomy, and by Easton and Watson, advising cecostomy or enterostomy, will be discussed in a later report. Here, too, the treatments should be grouped together, so that the patient may obtain rest and sleep between times. The importance of team-work with the senior attending surgeons in the management of these cases cannot be too strongly emphasized.

GROUP 5: ACUTE APPENDICITIS WITH ACUTE GENERALIZED FIBRINOPURULENT PERITONITIS

Definition.—Fortunately, only a few cases belong to this group. The condition represents the terminal stage of an uncontrolled, diffuse or spreading peritonitis. When one operates in such a case, the pathologic process presents itself about as follows: There is edema of the deeper portions of the rectus abdominis muscle or the transversalis abdominis muscle, or both. The properitoneal tissues and transversalis fascia are likewise edematous and congested. The parietal peritoneum is thickened because of marked edema, injection and deposition of fibrin; these fibrinous areas often represent sites of adhesions between the parietal peritoneum and the abdominal viscera. The latter are likewise intensely inflamed, and the serosa no longer retains its smooth glistening surface but now appears red, roughened and angry. Fibrinous plaques and adhesions are seen everywhere between adjacent structures, and if these are separated, there will remain a bleeding, torn, friable, shaggy surface. Not only is there pus containing flakes of fibrin throughout the peritoneal cavity, but in addition the many adhesions tend to enclose small collections of similar exudate. The pus may be thick or thin and pale or creamy or blood-tinged; it sometimes has a strong, foul odor, and on other occasions it may be relatively odorless; in a few instances it has been described as producing a peculiar fishy smell. The appendix may have completely sloughed away, and fragments of it are sometimes floating in the purulent exudate. Evidences of previous earlier abscess formation may still be seen, and in certain instances one can detect the point of rupture with subsequent spreading and ultimate fibrinopurulent peritonitis. The intestinal coils are considerably dilated, as a rule, and the mesentery exhibits marked thickening from edema and infection. The mesenteric serosa is extremely friable and bleeds if torn. The mesenteric blood vessels, especially the veins, may be undergoing extensive thrombosis. There is almost always considerable retroperitoneal cellulitis in the vicinity of the appendix.

Mortality Rate.—Of 25 patients with conditions of this type on whom operation was performed during the eighteen years of the present study,

only 3 survived, producing a total average mortality rate of 88 per cent. The figures in tables 13 and 14 speak for themselves, so that it is unnecessary to furnish mortality graphs.

TABLE 13.—*Annual Mortality Rates in Cases of Acute Appendicitis with Acute Progressive Fibrinopurulent Peritonitis at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916	0	0	0
1917	1	1	100
1918	0	0	0
1919	4	4	100
1920	1	1	100
1921	2	1	50
1922	1	1	100
1923	2	1	50
1924	2	2	100
1925	0	0	0
1926	0	0	0
1927	3	3	100
1928	2	2	100
1929	1	1	100
1930	2	2	100
1931	1	1	100
1932	0	0	0
1933	3	2	66
Total	25	22	88

TABLE 14.—*Progressive Five Year Average Mortality Rates in Cases of Acute Appendicitis with Acute Progressive Fibrinopurulent Peritonitis at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916–1920	6	6	100.0
1917–1921	8	7	87.5
1918–1922	8	7	87.5
1919–1923	10	8	80.0
1920–1924	8	6	75.0
1921–1925	7	5	71.42
1922–1926	5	4	80.0
1923–1927	7	6	85.71
1924–1928	7	7	100.0
1925–1929	6	6	100.0
1926–1930	8	8	100.0
1927–1931	9	9	100.0
1928–1932	6	6	100.0
1929–1933	7	6	85.71

Comparative Study.—Walker [15] reported a death rate of 33.3 per cent; Guerry [1] (personal series) gave the same figure, and Potter [14b] showed an average of 83.3 per cent (1920-1934, six cases with five deaths).

Analysis of Deaths.—The 22 patients who died were almost equally distributed between the two sexes, 12 being males. The average age was 41, and there were only 6 patients under 30 years. The youngest was 7. Here again the highest

incidence fell into the fifth, sixth and seventh decades. The duration of the present illness varied from two days to eleven days, but most of the patients had a history of illness of two, three or four days' duration. The average duration was three and eight-tenths days. Three patients had an existing infection of the upper respiratory tract at the time of their admission, and another had active pulmonary tuberculosis. One patient had undergone an operation for acute osteomyelitis five days before and two days later complained of cramps in the lower portion of the abdomen and vomiting. The symptoms and signs were quite confusing, and it was only after general abdominal tenderness and rigidity developed that an acute abdominal condition requiring operation was recognized. Interestingly enough, the culture from the peritoneal exudate grew a different organism from that of the previous acute infection of the bone. In most of the cases a general anesthetic was used, especially the combination of nitrous oxide and ether. Local anesthesia was used only twice, and 3 other patients received spinal anesthesia. The duration of the operation varied from twenty to forty minutes in the majority of instances. Appendectomy was performed in 14 cases and enterostomy or jejunostomy in 5. Cecostomy was performed once. The postoperative complications were almost always severe. The overwhelming nature of the infection, with toxemia, distention and paralytic ileus, served to terminate life within a few hours or sometimes in one, two or three days. Fourteen patients died from general sepsis; 9 had marked distention and a paralytic ileus. Severe shock developed in 8, and acute gastric dilatation occurred in 4. Pulmonary edema was a terminal complication in 4 cases and pneumonitis in 7 others. There was complete urinary suppression in 3. Two patients died on the operating table. Secondary peritoneal abscess is recorded only once, because almost all these patients died shortly after the operation. Only 4 lived longer than three days. One patient died on the fifth day from urinary suppression, and at that time the peritoneal infection seemed to be controlled. The second patient, a boy of 14, lived eighteen days; pertonitis had subsided, but he failed to cooperate, and his activities provoked disruption of the wound. This was followed by severe secondary hemorrhage, resulting in shock and death in spite of several transfusions. In a third patient, the stormy postoperative period of distention and gastric dilatation had been surmounted. The patient seemed to be recovering, but at the beginning of the second week high fever developed, and the blood culture yielded a pure growth of the colon bacillus. Death intervened on the thirteenth day. The fourth patient had survived the severe reaction of the first week only to have an ischiorectal abscess develop, which was incised and drained on the sixteenth day. Once again it appeared that he would recover, but on the twenty-fifth day there were signs of peritoneal abscess. It was drained through the old incision, but the patient failed to respond to supportive measures and died on the thirty-fourth day. He was 60 years old.

The treatment of these patients is extremely difficult because it requires careful judgment, based on experience. One must guard against too much therapy, yet it must be well directed against gastrointestinal, pulmonary and urinary complications. The proper choice of an anesthetic is extremely important here; perhaps spinal and local anesthesia could be employed a little oftener. Preoperative gastric lavage and clysis with saline solution before or during the operation are helpful in patients with marked distention and ileus. The operation should be done quickly, with the least possible trauma, and in most instances it is probably unwise to remove the appendix. One should be content to drain

the original focus or abscess and do no more. If the coils are markedly distended, jejunostomy or enterostomy can be quickly executed, and such a procedure should not be postponed until the patient is hopelessly beyond help. The postoperative care should consist of periods of adequate treatment, interspersed with several hours of sleep and rest; these sick patients cannot combat their infection without rest and plenty of it. Large amounts of fluids are most important, especially saline solution intravenously or subcutaneously, in order to supplant the loss of electrolytes incurred by the ileus and vomiting. The continuous intravenous drip is useful, but there is a real danger of giving too much, as this may precipitate acute pulmonary edema. The daily intake and output should be carefully recorded. The use of the nasal gavage tube will often prevent gastric dilatation resulting from regurgitation into the stomach of the contents of the duodenum and upper part of the intestine.[16a] Distention and paralytic ileus may be extremely difficult to handle, and the frequent use of abdominal poultices and the rectal tube, together with certain carminative enemas, may control it. Solution of pituitary or pitressin may be given with these treatments. Certainly there can be no danger of spreading the peritoneal infection any further. I have not been impressed with the effects of pitressin in cases of extensive peritoneal infection or of pronounced paralytic ileus. Transfusions and stimulants should be used before shock or collapse develops. The patients are probably more comfortable in a low or medium gatch position, and it is well to turn them at intervals as one means of preventing pneumonia and pressure sores. Hypertonic solution of dextrose (10 per cent) blood transfusions and hot colonic irrigations may be employed to combat renal suppression, although recently there is evidence at hand to indicate that saline solution is sometimes more efficacious than hypertonic dextrose solution. If the patient can be carried over this critical period, then one must prepare to meet other complications, such as secondary peritoneal abscesses, pylephlebitis, subphrenic and hepatic abscess, hemorrhage and the lighting up of some latent disease process, such as pulmonary tuberculosis. The surgeon, therefore, should always endeavor to maintain a clear mental picture of the various

16a. Since this paper was written, some encouraging results have been obtained in cases with severe peritonitis and paralytic ileus by the combined use of the nasal gavage tube together with intravenous drip or infusion of a solution of alcohol in aqueous sodium chloride solution, as recommended by Dr. Sterling Mueller at the Bellevue Hospital, New York. It is prepared by adding 100 cc. of 98 per cent alcohol to 900 cc. of physiologic solution of sodium chloride. The nasal tube is connected to a water-suction apparatus so as to exert slight, continuous suction. This treatment will probably cut down the indications for jejunostomy for paralytic ileus. In a few cases it resulted in a striking and dramatic change in the patient within from twelve to twenty-four hours. The use of this alcoholic solution in saline usually results in a local thrombosis of the vein.

disease processes that are taking place and to give them their proper interpretation. In this manner his treatment will be rational and well directed and should yield the maximum benefit to his patient. There are, in addition, certain other preventive measures which will be discussed a little later.

Cases of Survival.—The cases of the 3 patients who survived deserve brief mention. The first patient was a 12 year old boy with a history of illness of sixty hours' duration. A day after the onset of his illness, he took magnesium citrate. On admission, he looked acutely ill, and had a temperature of 103 F. and a pulse rate of 130. The abdomen was rigid throughout, and there was diffuse tenderness,

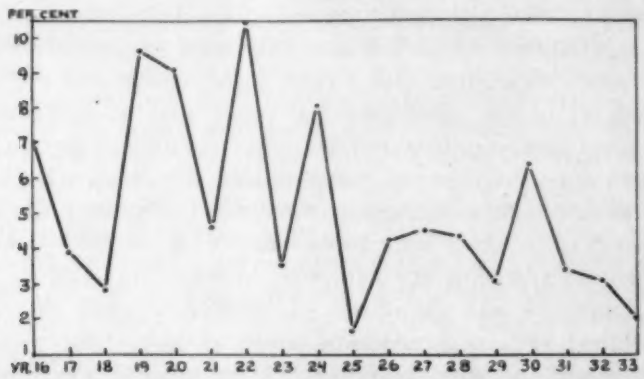


Chart 9.—Graph showing the total annual death rate from 1916 to 1933, inclusive, in 2,653 cases of acute appendicitis with its associated lesions. The total number of deaths was 135; the total mortality rate, 5.08 per cent.

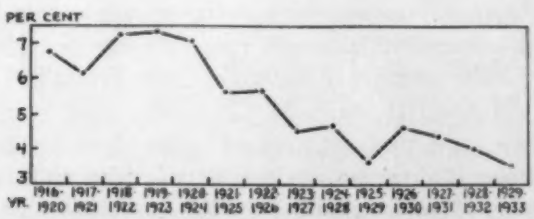


Chart 10.—Graph for comparison with that in chart 9, showing the total progressive five year average death rate from 1916 to 1933, inclusive, in the same group of cases of acute appendicitis with its associated lesions.

perhaps a little more marked in the right lower quadrant and the suprapubic region. Rectal examination revealed tenderness high up on the right side. The white blood cells numbered 10,200, with 76 per cent polymorphonuclears. The pathologic process at operation was typical of acute progressive fibrinopurulent peritonitis. The appendix was procecal and entirely gangrenous. Culture of the pus showed Bacillus acidi-lactici, Bacillus faecalis-alkaligenes, Streptococcus faecalis and a gram-negative bacillus. A soft rubber tube was placed in the pelvis, and a cigaret drain was carried to the ileocecal region and another to the right lumbar gutter. There was a moderate reaction but no distention and only slight vomiting. Aside from the use of a poultice and a rectal tube, no rectal treatment was given until the seventh day,

when the patient received an enema. Daily hypodermoclyses and infusions were administered. Not a single complication developed. The patient was out of bed on the eighteenth day after operation and went home on the twenty-third day. A three year follow-up study showed him to be in excellent condition, symptom-free, with a firm scar.

The second case was similar to the first. The boy was 12 years old and gave a history of illness of five days' duration. He also took a strong cathartic a day after the onset of symptoms, following which his mother gave his abdomen a massage! The pains were first in the lower portion of the abdomen; then they localized on the right side, and finally they became generalized. The boy looked acutely ill and had a temperature of 103.4 F. and a pulse rate of 120. There were general abdominal rigidity and tenderness as well as rebound tenderness. The white blood cells numbered 20,000 with 96 per cent polymorphonuclears. At operation the appendix was as large as a man's thumb, and the distal third was wrapped in omentum. The pathologic process was one of acute progressive fibrinopurulent peritonitis. The nonhemolytic streptococcus and Bacillus lactis-aerogenes were cultured from the pus. One soft rubber tube and a cigaret drain were placed in the pelvis, and another soft rubber tube was carried to the right lumbar gutter. The patient reacted well. There was no vomiting and practically no distention. The first rectal treatment was given on the third day. Hypodermoclysis and infusion were given daily. Signs of a residual abscess in the left lower quadrant developed on the sixteenth day, but it seemed to resolve five days later. The patient was out of bed on the twenty-first day and went home on the thirtieth day. He was last seen at the follow-up clinic nineteen months after his operation, in good condition, with no complaints and with a firm scar.

The third case was that of a 22 year old man, with a history of illness of four days' duration. Following the onset of generalized abdominal cramps, he purged himself with salts and then proceeded to take three successive doses of castor oil (including a double dose!). There was no nausea or vomiting. He returned to work on the third day of his illness and went to a baseball game on the day of admission. He then consumed a large supper, and only because he vomited about an hour later did his mother bring him to the hospital. He looked very ill. The temperature was 102.4 F., and the pulse rate, 130. The abdomen was boardlike, yet the patient did not complain of tenderness anywhere, even on deep palpation. There was also absence of rebound tenderness. There was slight tenderness on rectal examination, high up anteriorly in the midline. The white blood cells numbered 12,700, with 74 per cent polymorphonuclears. At operation, one recognized typical progressive fibrinopurulent peritonitis. The omentum had covered the appendix, which was completely gangrenous and had perforated. About 1½ quarts of pus was aspirated. Only one organism grew from the culture, Bacillus coli-communis. One cigaret drain was introduced into the pelvis and another at the site of the appendix. There was a rather marked reaction but no vomiting. Pitressin, with the use of a poultice and a rectal tube, was given every four hours for three days, and this served to control the slight distention. Colonic lavages were administered a few times, and the patient received hypodermoclysis and infusion daily. The temperature was normal on the eighth day. All drains were removed on the eleventh day. On the thirty-third day he was out of bed, and he went home three days later. It is only five months since his operation, but so far the results have been entirely satisfactory.

The reader will observe that the patients who survived were males —2 boys and a young man. All took cathartics. In two instances the

total white cell count was relatively low, and the differential count was only slightly elevated. All patients had a surprisingly smooth postoperative course. The follow-up study, to date, has revealed most satisfactory results.

TABLE 15.—*Total Annual Mortality Rates in All Cases of Acute Appendicitis and Associated Lesions at the Presbyterian Hospital*

Year	Number of Cases	Number of Deaths	Mortality, per Cent
1916	128	9	7.03
1917	128	5	3.90
1918	135	4	2.88
1919	133	13	9.77
1920	155	14	9.03
1921	150	7	4.66
1922	142	15	10.56
1923	167	6	3.59
1924	147	12	8.16
1925	122	2	1.63
1926	139	6	4.31
1927	131	6	4.58
1928	134	6	4.47
1929	159	5	3.14
1930	154	10	6.49
1931	149	5	3.35
1932	188	6	3.19
1933	192	4	2.08
Total	2,653	135	5.08

TABLE 16.—*Progressive Five Year Average Mortality Rates in All Cases of Acute Appendicitis and Associated Lesions at the Presbyterian Hospital*

Years	Number of Cases	Number of Deaths	Mortality, per Cent
1916–1920	679	45	6.62
1917–1921	701	43	6.13
1918–1922	715	53	7.41
1919–1923	747	55	7.36
1920–1924	761	54	7.09
1921–1925	728	42	5.76
1922–1926	717	41	5.71
1923–1927	706	32	4.53
1924–1928	673	32	4.75
1925–1929	686	25	3.64
1926–1930	717	33	4.60
1927–1931	727	32	4.40
1928–1932	784	32	4.08
1929–1933	842	30	3.56

TOTAL MORTALITY RATE FOR GROUPS 1 TO 5

Table 15 and charts 9 and 10 are probably the most interesting of those shown, because they represent the total mortality rate in cases of acute appendicitis and its associated lesions. Whereas there is a certain factor of error in the data in the previous tables (as mentioned in the beginning of this report), the figures presented in tables 15 and 16

can be considered accurate. The total death rate has diminished in the past three years, and for 1933 (2.08 per cent) it is close to the record low level of 1925 (1.63 per cent). The progressive five year average of the total annual death rate shows a slight but definite decrease in the past seven periods as compared to the seven earlier ones (chart 10). The most recent one (1929 through 1933) discloses a record low level

TABLE 17.—*Total Mortality Rates in Series of Cases of Acute Appendicitis and Associated Lesions Reported in the Literature*

Year of Publication	Author	Place	Number of Cases	Period Studied	Mortality, per Cent
1923	Lower and Jones: J. A. M. A. **81**: 629 (Aug. 25) 1923	Cleveland	2,067	Not stated	3.1
1924	Deaver and Magoun: Ann. Surg. **79**: 854, 1924	Lankenau Hospital, Philadelphia	5,488	1900-1920	5.0
1925	Adams [7]	London Hospital	1,677	1919-1923	5.8
1925	Love [8]	St. Thomas' Hospital	901	1919-1923	8.4
1927	Colp [9]	Mount Sinai Hospital, New York	2,841	1916-1926	5.2
1928	Weeden: Ann. Surg. **88**: 76, 1928	New York Hospital	1,588	1914-1927	4.9
1931	Miller: J. Coll. Surg. Australasia **3**: 40, 1931	Touro Infirmary and Charity Hospital, New Orleans	2,415	1924-1929	9.9
1931	McDonald: Minnesota Med. **14**: 212, 1931	St. Mary's and St. Luke's hospitals, Duluth, Minn.	1,574	1925-1930	4.5
1932	Boland: J. A. M. A. **99**: 443 (Aug. 6) 1932	Atlanta hospitals	4,270	1927-1932	4.4
1932	Raynor: Proc. Roy. Soc. Med. **26**: 181, 1932	Manchester Royal Infirmary	1,877	1928-1931	5.0
1932	Colt and Morrison: Brit. J. Surg. **20**: 197, 1932	Aberdeen Royal Infirmary	1,349	1911-1930	5.5
1933	Bauer [11]	General Hospital, Malmö, Sweden	5,208	1903-1930	2.3
1933	Walker [12]	Boston City Hospital	2,106	1927-1930	5.8
1934	Bower: J. A. M. A. **102**: 813 (March 17) 1934	Philadelphia hospitals	14,904	1928-1932	4.79
1934	Nuttall: Brit. J. Surg. **21**: 411, 1934	Liverpool Royal Infirmary	551	Not stated	2.5
1934	Garlock: Am. J. Surg. **23**: 248, 1934	New York Hospital, II Division	1,188	1921-July 1932	4.7
1934	Keyes: Ann. Surg. **99**: 47, 1934	Barnes and St. Louis Children's hospitals	1,099	1915-1932	5.0
1934	Quain: Arch. Surg. **28**: 782 (April) 1934	Bismarck, N. D.	1,000	Sept. 1927 to Oct. 1932	3.8

of 3.56 per cent, which is 1.5 per cent lower than that of the complete total of 2,653 cases with a mortality of 5.08 per cent.

This compares favorably with total mortality statistics in other hospitals (table 17). Some of the personal series had lower rates, for example: Guerry,[1] 1.1 per cent; Stoney,[4] 2.5 per cent; Hawe,[6] 4 per cent; Dixon,[8] 1.52 per cent; F. Angel and E. Angel,[17] 2.2 per cent, and Greenwood,[18] 0 per cent; (Greenwood cited a series of 206 cases covering

17. Angel, F., and Angel, E.: South. Med. & Surg. **95**:87 (Feb.) 1933.
18. Greenwood, H. H.: Lancet **1**:973 (May 11) 1929.

a two year period only). Other authors reporting on personal series, or those representing cases observed by a small limited group of surgeons, submit mortality rates close to those of the Presbyterian Hospital: Finney,[2] 4.92 per cent; a few others give a higher rate, as Eliason and Ferguson,[19] 5.3 per cent and Warren,[20] 6 per cent.

Chart 11 shows the curves of the five year averages in groups 1 to 4 and also that of the total mortality curve from 1916 to 1933 inclusive, thus enabling the reader to appreciate the relationships of the different groups to each other.

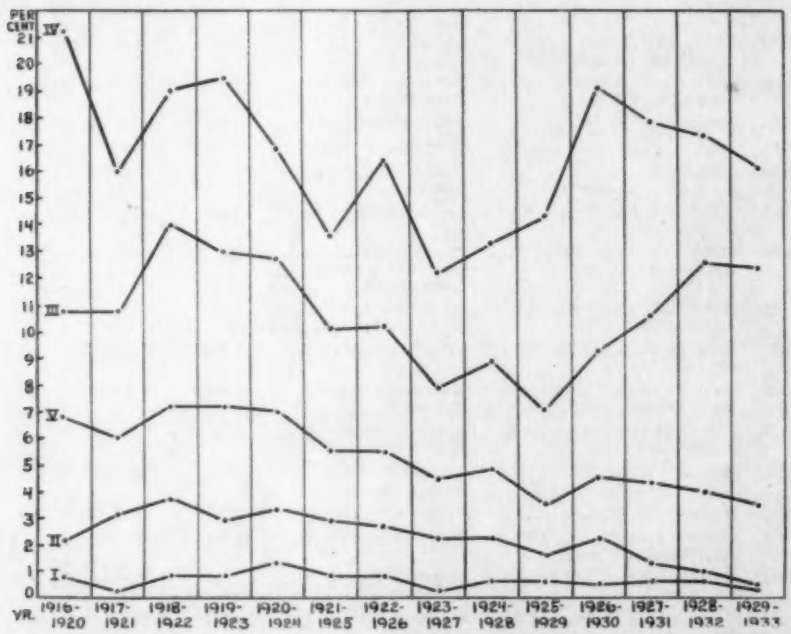


Chart 11.—Graph showing the progressive five year average mortality curve from 1916 to 1933 for groups 1 to 4 and also the total progressive five year average mortality rate in all cases of acute appendicitis and its associated lesions. Curve I represents the cases of acute appendicitis; II, the cases of acute appendicitis with acute local peritonitis; III, the cases of acute appendicitis with peritoneal abscess; IV, the cases of acute appendicitis with acute diffuse peritonitis, and V, the total progressive five year average mortality rate of all cases of acute appendicitis and its associated lesions.

COMMENT

The general mortality rate for acute appendicitis and its associated lesions at the Presbyterian Hospital has definitely declined. In separating

19. Eliason, E. L., and Ferguson, L. K.: Ann. Surg. **88**:65 (July) 1928.
20. Warren, R.: Lancet **2**:16 (July 6) 1929.

the various groups, the death rate from simple acute appendicitis remains at a fairly constant low level. The mortality from acute appendicitis with acute local peritonitis has shown a distinct decline. On the other hand, the mortality rate has increased in the next two groups, cases of acute appendicitis with abscess and of acute appendicitis with acute diffuse peritonitis. To be sure, the recent annual death rate in both groups shows a distinct decline, but the five year average leaves no doubt as to its increase compared to former periods. The death rate from acute appendicitis with acute fibrinopurulent peritonitis remains consistently high, but this should not discourage one from enlisting one's greatest efforts in an attempt to save patients with this condition. The very fact that 3 actually did survive should encourage one to carry on a strong battle for their lives.

Discussions on the possible means of reducing the mortality in the various groups (except group 4, acute appendicitis with acute diffuse peritonitis, which will appear in a separate paper) have already been given, and they need not be repeated here. They may merit consideration on the part of the surgeon who is ever anxious to better his results.

There should be perfect team-work on the part of the hospital staff in the treatment of patients with peritonitis and abscess. The older experienced men should guide and direct the younger attending surgeons and house staff officers in handling these cases, because they demand the most careful, balanced and mature judgment; the problems and complications may be extremely difficult to comprehend and to combat.

One phase in the possible reduction of the mortality rate does deserve discussion here, namely, the problem of prevention. This directly concerns the laity as well as the medical profession and is largely a public health problem. A cursory glance at the tables will show at once that the mortality rate in cases of simple acute appendicitis is very low, but as soon as perforation takes place, with spreading peritonitis, the mortality rate is high. The public should learn that the early diagnosis of acute appendicitis followed by early operation by a competent surgeon at a well equipped hospital is attended by an extremely low mortality rate. There are certain factors which may interfere with this:

1. Certain fears and superstitions still exist in the minds of many that to enter a hospital and undergo an operation is an acknowledgement of defeat and can result only in certain death. The problem is often exasperating, and the practitioner can do nothing but observe the chances of recovery gradually diminishing with the advance of time. When consent to operation is finally given, it may be too late.

2. Mothers still give their children cathartics for abdominal cramps and pains. The public health service and pediatricians should carry on a vigorous campaign against the indiscriminate use of such medicine. It is simpler and safer to give an enema instead or, at least, to give a cathartic only after examination by a physician.

3. The indiscriminate dispensing of purgatives and laxatives by the pharmacist is a factor. Persons constantly enter a drug store and request some medicine, to be recommended by the druggist, for abdominal pain. This practice is becoming increasingly widespread, especially in recent years when many persons have been unable to consult a physician.

4. Most persons associate appendicitis with pain on the right side. Few, if any, realize that an acute attack starts, as a rule, with generalized (or epigastric or periumbilical) abdominal cramplike pain. Consequently they believe they have an "upset stomach" or "indigestion," and resort to the time-honored cathartic. Only later, when the pain localizes in the right side (and this does not always happen), do they call a physician. Meanwhile, valuable time has been lost. It is most important to educate the public, therefore, that pain on the right side develops only later in cases of acute appendicitis, and they should learn to appreciate the factor of delay in its relation to mortality.

5. The physician himself may be unable to recognize some of the early atypical cases of acute appendicitis. If that is the case he should observe the patient frequently, perhaps every two hours, until more definite signs develop. He should remember never to give opiates for abdominal pain until he can make a definite diagnosis. In the event of continued doubt he should always request a consultation even though it may touch his pride a bit; surely a doctor's pride cannot compare with a patient's life. He must learn to regard with suspicion any abdominal pain that persists over six hours.

6. The practitioner knows only too well that a certain number of cases of acute appendicitis will subside with morphine, rest and the use of an ice bag. He institutes this therapy for twenty-four hours (the most important factor in reducing the mortality is therefore lost) and returns the next day. If the patient is better, the therapy is continued and recovery probably takes place. The physician is considered a miracle man; he saved his patient from an operation and "froze" out the appendix. But what if the patient is worse or only about the same? The physician will decide to send him to the hospital or else will determine to watch him for another twenty-four hours. In either case the damage has been done. The patient no longer has a localizing process, but probably spreading peritonitis, due to perforation or gangrene of the appendix. The physician has done "his best;" he

has tried to save his patient from an operation and, in so doing, the patient's life has been jeopardized. So the patient goes to the hospital and either has a long stormy course, which may be of great economic loss, or else dies. The result is that the hospital and the surgeon are blamed, and the practitioner is completely exonerated. From the standpoint of economy alone the public should learn that the conservative and economic treatment of acute appendicitis is early prompt operation.

7. In times like the present one, the practitioner may choose to operate on the patient himself, or the family may request it. He is the "occasional operator." He frequently lacks operating privileges in some of the larger, better equipped hospitals. So the patient may be taken to a smaller private hospital. At operation, the physician may encounter a situation with which he is entirely unable to cope. Appendectomy may be the simplest or the most difficult operative procedure. He lacks the judgment and skill and experience in making important decisions and in executing technical maneuvers. Dire events then take place. One complication follows another. The hospital may be inadequately equipped to meet the demands for certain important treatments and emergencies. A death results which might have been prevented in proper hands. The public must be taught the importance of receiving skilled care in a well equipped hospital.

8. Instruction to high school and university students, as recommended by the Philadelphia group, is an excellent plan, because it is known that the highest incidence of appendicitis occurs between the ages of 13 and 30. Such a policy could be introduced into large business concerns and corporations. The program to reduce the mortality from appendicitis in Philadelphia is being conducted so successfully that there has been a real diminution in fatalities, and consequently in the death rate, in the past five years.

These are not original or new ideas. They have been repeatedly emphasized in the literature. It is the real duty of the public health service, of the county, state and national medical societies and of the insurance companies to present these facts in an intelligent dispassionate manner before the public. It would be extremely unwise to employ alarmist tactics because many persons with vague pains on the right side would soon find their way to the operating table to undergo an utterly useless and unnecessary appendectomy. One is confronted, therefore, with the present danger of insufficient public education on the one hand and the treachery of overenthusiastic propaganda on the other. Concerted, well directed, organized action could be made to follow the middle course.

SUMMARY

The total mortality rate from acute appendicitis at the Presbyterian Hospital from 1916 to 1933, inclusive, is 5.08 per cent, or 135 deaths

in 2,653 cases. The total death rate for each of the five groups is as follows: acute appendicitis, 0.59 per cent; acute appendicitis with acute local peritonitis, 1.9 per cent; acute appendicitis with peritoneal abscess, 10.5 per cent; acute appendicitis with acute diffuse (diffusing, spreading) peritonitis, 17.02 per cent, and acute appendicitis with progressive fibrinopurulent peritonitis, 88 per cent.

The curve of the progressive five year average of total deaths from acute appendicitis in all groups (from 1 to 5) shows a moderate, general decrease (chart 10). The five year average mortality curves of groups 1 and 2 indicate a decrease, but groups 3 and 4 disclose a definite, alarming increase, compared to earlier five year periods (chart 11).

An analysis of the deaths and a discussion of certain means of preoperative, operative and postoperative preventive measures have been presented for each of the groups (except group 4, which will be reported on in detail in another paper).

There is need for proper public education in the attempt to effect a general reduction in the mortality rate of acute appendicitis. The surgeon and the practitioner must also learn to recognize the atypical case early.

The death rate from acute appendicitis and its associated lesions can, and must, be reduced.

POSTANESTHETIC HEADACHE

P. W. HARRISON, M.D.
MUSCAT, ARABIA

There are three possible methods of anesthesia. One can anesthetize sensory nerve endings so that their stimulation gives rise to no afferent impulses. One can interrupt the transmission of afferent impulses by anesthetizing the nerve trunks which carry them. And finally one can stupefy the entire organism so that afferent impulses fail to register on arrival at the higher centers.

Anesthetization of the sensory endings has the advantage of safety, but for the patient it makes surgical procedure a harrowing experience, and the capacity of the tissues to heal is lowered by the necessary infiltration with fluid. The disadvantages of a general intoxication so profound that the higher centers are incapable of reacting to afferent impulses require no discussion.

Theoretically conduction anesthesia is much the best. There is no general intoxication, and no injury to the tissues involved in the operation. Spinal anesthesia is the ideal conduction method. In the subarachnoid space the sensory nerves are assembled in a beautifully convenient way. If one were able to avoid the complicating factors, spinal anesthesia would long since have displaced every other method.

The complicating factors have not been avoided. Cerebral anemia and respiratory paralysis have caused death in many cases, and at the moment spinal anesthesia is the most dangerous method in common use. But the discussion of these catastrophes has obscured the fact that for operations below the umbilicus spinal anesthesia, far from being excessively dangerous, is perhaps the safest method, and in comfort to the patient and convenience to the surgeon is almost ideal. Anesthetization of the point of spinal puncture makes the procedure practically painless for the patient, and recollection that heavy solutions gravitate downward makes the method safe. If the Trendelenburg position is wanted a heavy solution must not be used, and procaine crystals dissolved in spinal fluid form a heavy solution.

But an anesthetic must do more than furnish a satisfactory period for the execution of some surgical procedure. It must leave the patient undisturbed and comfortable after the operation is over. Spinal anesthesia has a bad record on this point. Postanesthetic headache has occurred frequently, and sometimes has been very severe. The dangers from a poorly given general anesthesia are much more serious than the discomfort of an occasional severe headache, but when the comparison

is made with the effects of ether expertly given, the headache may be sufficient to throw the whole method of spinal anesthesia into the discard. On the other hand, if the postanesthetic headache can be eliminated, for operations below the umbilicus spinal anesthesia stands in a class by itself.

Not much progress has been made in dealing with the problem of headache. It is difficult to investigate. There is a widespread impression that continued leakage from the puncture hole sometimes occurs and that this leakage by reducing the pressure within the subarachnoid space causes the headache. This may have been true once, when shockingly coarse needles were used even for routine diagnostic punctures. Fine delicate needles are used universally now, and the headache remains. The holes made by such delicate needles must be studied with a magnifying glass. They are tiny slits the edges of which automatically approximate on withdrawal of the needle, and it is impossible to believe that they permit any significant leakage, at least for any great length of time.

Regarding some other possible causes of headache there is more certainty. Postanesthetic headache will occur if the injected solution is irritating. Wayne Babcock,[1] the dean of the spinal anesthetic faculty, reported his experience with defective distilled water. Headache occurred frequently until this fault was corrected. I have seen the same thing in Muscat. Old rubber tubing in the distilling plant caused postanesthetic headache which for frequency and severity I have not seen equaled since. The fault was corrected, and the incidence of headache dropped to its former figure. A few months ago the solution which had caused almost no trouble in Muscat was reported as causing many headaches in a neighboring clinic. I found that spinal puncture was being made through skin dripping with tincture of iodine.

This matter of the irritating character of the anesthetic has not received the attention that it deserves. Any one of several factors can make such a solution irritating. In the first place, it may be irritating because of the chemicals it contains. Stovaine has enjoyed a great vogue as a spinal anesthetic, but it is highly unsuitable, being much more irritating than procaine hydrochloride.[2] In the second place, an acid solution is irritating. Any solution injected into the subarachnoid space should have a p_H above 7 and not below. That was the fault of the solution which caused so many headaches in Muscat. It was strongly acid. In the third place, a solution which is either hypertonic or hypotonic is irritating. That is probably the reason that procaine

1. Babcock, Wayne: Surg., Gynec. & Obst. **59**:94 (July) 1934.

2. Braun, H.: Lokalanesthesie, ed. 3, Leipzig, Johann Ambrosius Barth, 1914, p. 133.

crystals dissolved in spinal fluid cause so much headache. Such a solution is strongly hypertonic and to that degree is irritating. In the cat at least the matter of isotonicity is of great importance. In that animal the exposed cervical cord can be edematized with Ringer's solution with no disturbance at all, but if the Ringer's solution is diluted with distilled water the cat dies within a few seconds. Finally, the temperature of the injected solution is important. There is no question that the injection of a cold solution into the subarachnoid space is irritating and harmful.[3]

Besides these positive characteristics of injected fluids it is to be remembered that solutions brought into prolonged contact with the tissues of the central nervous system may be irritating because of what they lack. The most common clinical demonstration of this is the violent headache which follows the introduction of air into the cerebrospinal space in ventriculography.[4] As it is saturated almost immediately by water vapor, it is impossible to think of the air as an irritant per se, but by virtue of what it keeps away from the tissues, in practical results it is an exceedingly severe irritant. Weed,[3] studying the reactions of the meninges in cats, found that the lack of calcium has a similar result. Any solution which lacks a trace of that element is irritating and will cause the death of the animal in a few hours.

The first step of the present investigation was the development of an anesthetic solution as nearly as possible free from irritating properties. Procaine hydrochloride is the least irritating of suitable drugs. A solution of procaine hydrochloride of a concentration of 5.48 per cent is isotonic. I have used various combinations of procaine hydrochloride with dextrose and saccharose in order to increase the viscosity of the solution, but so far as my experience goes a simple solution of procaine hydrochloride of a strength from 5 to 5.5 per cent is just as good as any of the combinations. To such a solution calcium chloride is added in a quantity sufficient to make its strength 0.024 per cent, this figure being taken from Weed's work on cats. In the winter this solution is warmed to body temperature before injection. In the summer Muscat is so warm that this point requires no attention. The p_H of the solution is set between 7 and 7.2.[5]

---

3. Weed, L. H., and Wegeforth, P.: J. Pharmacol. & Exper. Therap. **13**:317, 1919.

4. Sachs, Ernest: The Diagnosis and Treatment of Brain Tumors, St. Louis, C. V. Mosby Company, 1931, pp. 342 and 123.

5. Dr. W. C. Harden of Baltimore worked out the details of this solution. It was the hope originally that we could work together on a much more thorough investigation of the problems of spinal anesthesia, the chemical problems as well as the clinical ones, but Muscat and Baltimore are too far apart for that to be practicable.

The limitations of such a solution are obvious. It will afford an anesthesia of an hour to an hour and a quarter but not more. It is a heavy solution, as indeed any effective solution of procaine hydrochloride must be unless diluted with something like alcohol. Therefore it cannot be used with the patient in the Trendelenburg position and is unsuitable for operations in the upper portion of the abdomen, when it is imperative that the patient be in the Trendelenburg position to maintain the blood supply to the brain and usually for the actual operative procedure as well. In Muscat nupercaine is used for such operations.

With the introduction of this nonirritating solution severe postanesthetic headache has disappeared, and the postoperative comfort of the patients is gratifying. The clinical impression is that when this solution can be used the problem of spinal headache has been disposed of.

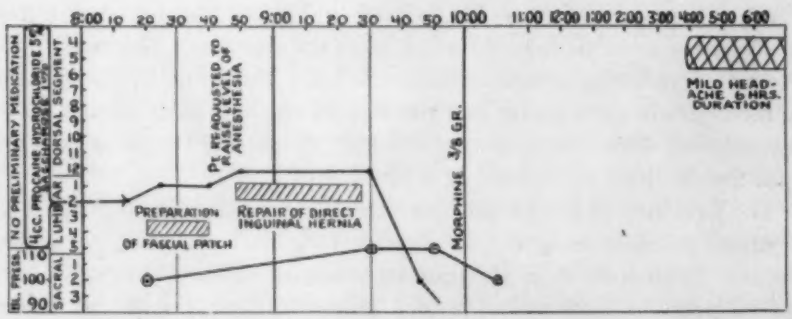

8:00 10 20 30 40 50 9:00 10 20 30 40 50 10:00 11:00 12:00 1:00 2:00 3:00 4:00 5:00 6:00
BL. PRES.
NO PRELIMINARY MEDICATION
4 CC. PROCAINE HYDROCHLORIDE 5%
DORSAL SEGMENT
LUMBAR
SACRAL
PT. READJUSTED TO RAISE LEVEL OF ANESTHESIA
PREPARATION OF FASCIAL PATCH
REPAIR OF DIRECT INGUINAL HERNIA
MORPHINE 3/8 GR.
MILD HEADACHE, 6 HRS. DURATION

Fig. 1.—A clinical picture confused by morphine given postoperatively to a high-strung Persian aged 32. The headache occurred six hours after the disappearance of the anesthesia and continued until the patient went to sleep at 10:00 p. m. Headache was not present on the following morning. The systolic blood pressure is indicated by the circles. The height of the anesthesia in segments is indicated by the solid dots. The arrow shows the level at which the anesthetic was introduced and the time of its introduction. The shaded areas indicate the operations, and the cross-hatched area, the headache.

Unfortunately this impression is not entirely correct. Questioning shows that many patients suffer from headache which is too mild to cause complaint. Moreover, they are comfortable only if they remain recumbent. Those who sit up and walk around after the operation frequently suffer from headache, which may be very severe and persistent. This was a disappointment, for in Arabia operations must be performed on many patients who wish to walk home afterward, and no method of anesthesia can be regarded as satisfactory unless it makes this possible.

The study of this form of headache offers certain difficulties. Postoperative medication can mask such a headache. Figure 1 is the chart for such a case. Various causes may result in headache quite uncon-

nected with the anesthesia. But in spite of the confusing factors, study of this type of headache has yielded certain results. In the first place, no matter what area is anesthetized or for how long, it is always the head that aches. The localization of the discomfort is vague and inconstant, even in the same patient, but there is never any doubt as to its being in the head. I see no way to interpret this except as a disturbance of the hydrostatics of the cerebrospinal system.

The second point of interest which emerges is the precise uniformity of the time of appearance. I have observed cases in which anesthesia

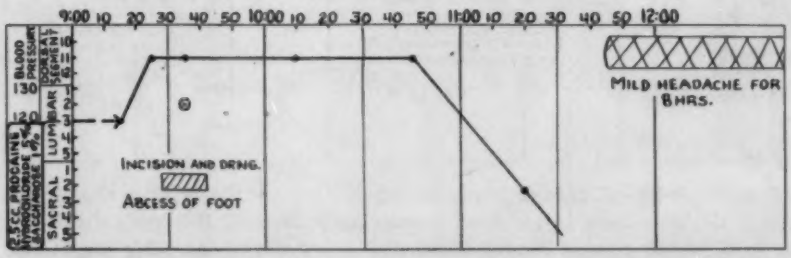

9:00 10 20 30 40 50 10:00 10 20 30 40 50 11:00 10 20 30 40 50 12:00
BLOOD PRESSURE
130
120
SACRAL
LUMBAR
INCISION AND DRNG.
ABCESS OF FOOT
MILD HEADACHE FOR 8 HRS.

Fig. 2.—A typical headache appearing immediately after disappearance of the anesthesia in an Arab Bedouin aged 35. He was up and about after the operation. He would probably have felt no headache if he had remained recumbent. His general condition was good.

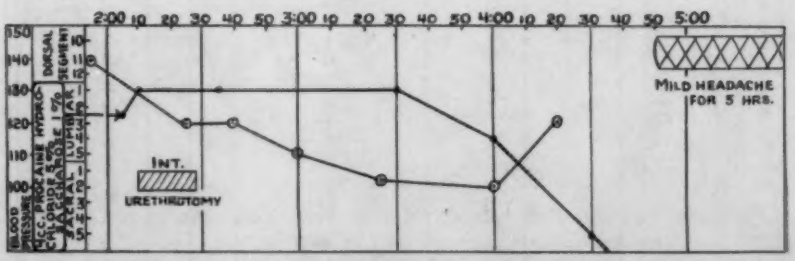


Fig. 3.—A typical case, with headache appearing immediately after the disappearance of the anesthesia. The patient was in good general condition.

with nupercaine lasted six and even eight hours, but I have never observed a case in which headache appeared during the course of the anesthesia. It always starts a short time after the disappearance of the anesthesia. This is a uniform finding whether the anesthesia lasts for thirty minutes or eight hours and whether it involves the fourth and fifth sacral segments only, as is usual in cases in which hemorrhoidectomy is performed, or extends to the third dorsal segment, as is required for a splenectomy. Figures 2, 3 and 4 illustrate this uniformity of appearance under varying circumstances.

I observe an occasional case in which no headache is felt but in which at the time when headache would occur other sequelae are experienced. Figures 5 and 6 are charts for such cases.

The uniform appearance of the headache immediately after the disappearance of the anesthesia strongly suggests that there is a vasomotor reaction. Spinal anesthesia abolishes the tone of the blood vessels in the extremities, a fact used in the study of Buerger's disease and allied

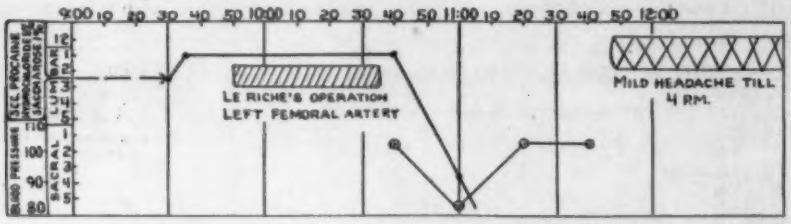

9:00 10 20 30 40 50 10:00 10 20 30 40 50 11:00 10 20 30 40 50 12:00
0.5% PROCAINE HYDROCHLORIDE (SACHAROSE) 1%
DORSAL
LUMBAR
SACRAL
BLOOD PRESSURE
LE RICHE'S OPERATION
LEFT FEMORAL ARTERY
MILD HEADACHE TILL
4 P.M.

Fig. 4.—A typical case of headache appearing immediately after the disappearance of the anesthesia in an Arab woman aged 30 with Buerger's disease. She was in very bad general condition and died a few days later, after amputation of the leg.

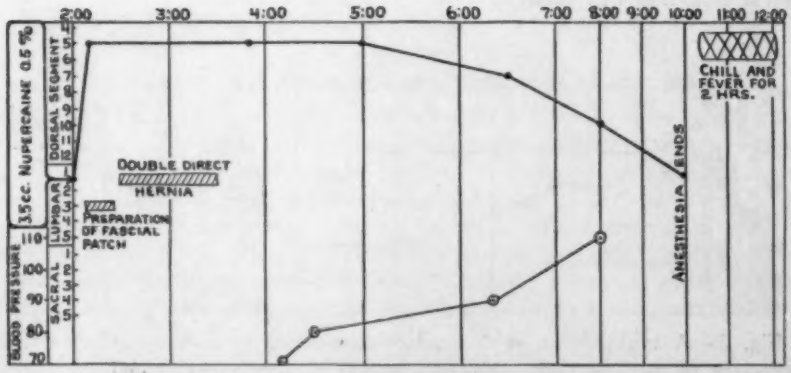


Fig. 5.—A case of prolonged nupercaine anesthesia in a Persian stoic aged 70. Even after such a long anesthesia the postanesthetic disturbance appeared immediately after the disappearance of the anesthesia. No headache was experienced in this instance, but fever was ushered in by a mild chill and lasted two hours.

troubles. It would seem certain that a similar result must be produced on the vessels of the meninges and the cord and that throughout the time of spinal anesthesia the vessels are toneless and dilated over the area bathed by the anesthetic solution. On the disappearance of the anesthesia vasomotor control of these vessels is reestablished, and at this point the headache appears.

This reestablishment of vasomotor control must inevitably involve some disturbance in cerebrospinal hydrostatics, for with it must come a shrinkage in the volume of the vessels and thereby a slight increase in the space to be occupied by the cerebrospinal fluid. With this must occur a slight drop in the intra-arachnoid pressure. The obvious objection to this analysis is that the changes in volume accompanying this reestablishment of vasomotor tone must be exceedingly small, and a priori one would suppose that such minute changes could be easily compensated for by the elasticity of the dural sac. Moreover, even in the cat the cerebrospinal fluid is secreted at a rate of about 12 cc. a day.[6] In man the figure must be much higher. Any deficit in cerebrospinal

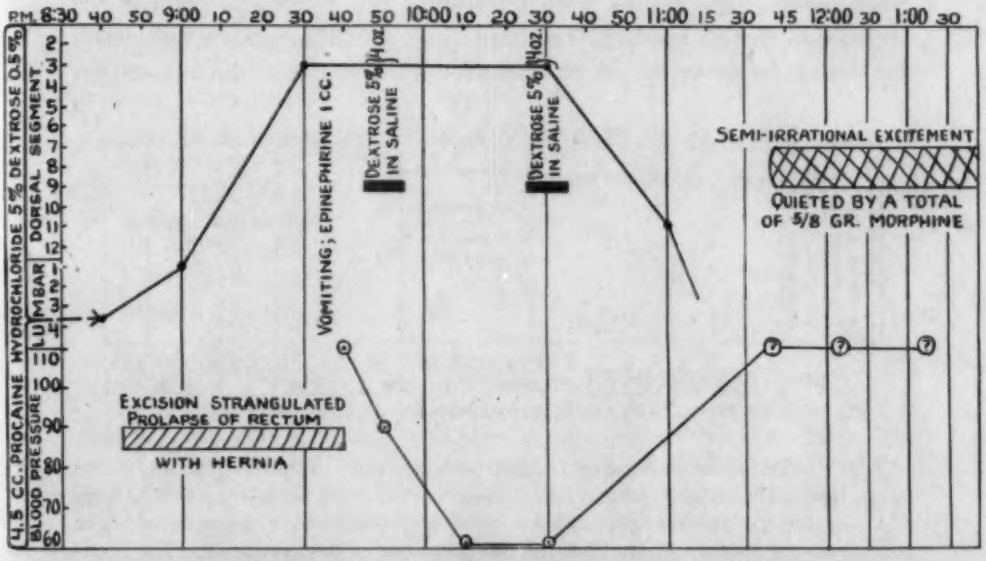

P.M. 8:30 40 50 9:00 10 20 30 40 50 10:00 10 20 30 40 50 11:00 15 30 45 12:00 30 1:00 30
4.5 CC. PROCAINE HYDROCHLORIDE 5% DEXTROSE 0.5%
BLOOD PRESSURE
MM. HG.
DORSAL SEGMENT
VOMITING; EPINEPHRINE 1 CC.
DEXTROSE 5% IN SALINE
DEXTROSE 5% IN SALINE
SEMI-IRRATIONAL EXCITEMENT
QUIETED BY A TOTAL OF 5/8 GR. MORPHINE
EXCISION STRANGULATED PROLAPSE OF RECTUM
WITH HERNIA

Fig. 6.—The patient, a Beloochi aged 50 of somewhat unstable mentality, was in a very toxic condition. His position on the table was disarranged and resulted in anesthesia of the upper segment. Artificial respiration was administered once and epinephrine once. Dextrose solution was given twice, making a total of 8 ounces. No headache appeared, but a period of irrational excitement followed immediately after the disappearance of the anesthesia. The patient spent this period walking about in the compound of the hospital.

fluid could be made up in a few hours at the longest, whereas the headache sometimes lasts for days.

This would seem to necessitate the conclusion that even though the inception of a postanesthetic headache may be due to a slight drop in intra-arachnoid pressure accompanying the reestablishment of tone in the previously anesthetized vessels, its continuance must be due either to a

6. Weed, Lewis: Personal communication to the author.

diminished secretion of cerebrospinal fluid or to its accelerated absorption from the subarachnoid space. The fluid is secreted by the choroid plexuses, and they are far removed from the influence of the injected anesthetic. That would seem to leave as the one remaining obvious possibility an accelerated absorption of the cerebrospinal fluid and, resulting from that, a persisting condition of abnormally low intra-arachnoid pressure.

It seemed possible to test some points in this analysis, and for this purpose we had made up ampules of the nonirritating solution previously described, to which had been added minute doses of ephedrine (1/25 grain [0.0026 Gm.]) and epinephrine (1/8 minim [0.0124 cc.]) respectively. The results were interesting and seemed to confirm the impression that in headache resulting from spinal anesthesia more than one factor is involved. With these solutions headache did not appear

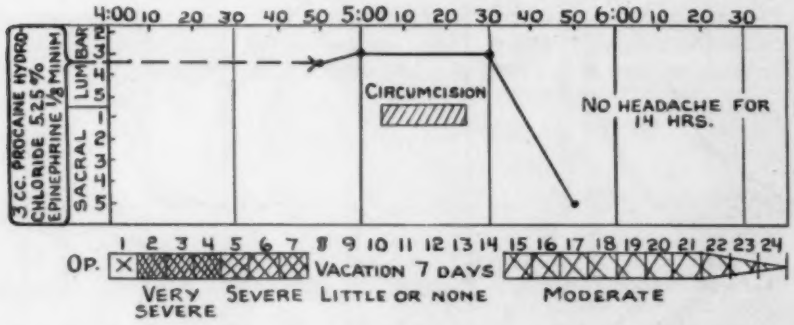

4:00 10 20 30 40 50 5:00 10 20 30 40 50 6:00 10 20 30
3 CC. PROCAINE HYDRO-CHLORIDE 5.25 % EPINEPHRINE 1/8 MINIM
LUMBAR
SACRAL
CIRCUMCISION
NO HEADACHE FOR 14 HRS.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
OP.
VACATION 7 DAYS
VERY SEVERE
SEVERE
LITTLE OR NONE
MODERATE

Fig. 7.—The delay in onset of headache when a trace of epinephrine was added to the injected anesthetic occurred in a large powerful Portuguese aged 40. This is a uniform result after the addition of either ephedrine or epinephrine. The onset occurred fourteen hours after the disappearance of the anesthesia. The headache was mild for one day, and was felt only when the patient shook his head. The headache was very severe for the following three days and fairly severe for the next three. The patient kept steadily at work in his office. A vacation of a week brought much relief, but the headache recurred when he resumed work and persisted for another week, after which it gradually disappeared. Cases of ambulant patients such as this are the severest test of spinal anesthesia. This man would have been far better off to have undergone short general anesthesia for such a minor operation as circumcision.

immediately after the disappearance of the anesthesia but followed a period in which the patient was free from all complaint. This free period usually lasted about twelve hours, though it was sometimes much longer. Unfortunately when the headache appeared it seemed to be undiminished in severity, i. e., negligible if the patient remained recumbent but sometimes severe if he walked about. Figure 7 is a good illustration.

I experimented with the intravenous injection of distilled water in the hope that an abnormal amount of water in the blood might stop excessive absorption of cerebrospinal fluid into the blood stream. The distilled water probably does have that effect, at least for a time, but it

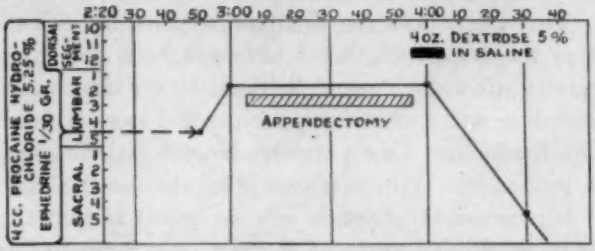


Fig. 8.—The patient, an Arab aged 30, gave a typical reaction following an intravenous infusion of dextrose as a prophylactic against headache. No headache occurred at any time.

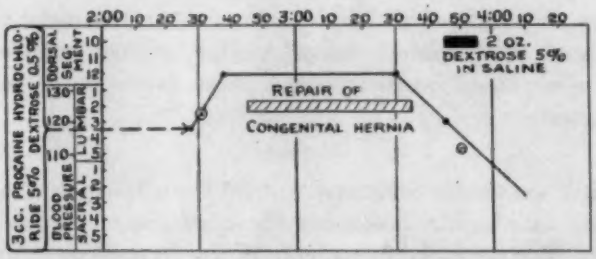


Fig. 9.—A typical reaction following prophylactic infusion of dextrose solution. The patient was an Arab boy of 10 years in poor general condition. He was put under no restraint, and sat up a large part of the time. No headache occurred at any time. He was able to sit up immediately after the operation.

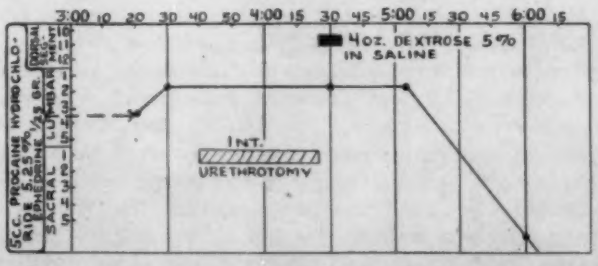


Fig. 10.—The maximum degree of headache following a prophylactic injection of a solution of dextrose. The patient received 5 cc. of a 5 per cent solution of procaine hydrochloride, which is the maximum dose in the Muscat clinic. He was up and about immediately following the operation. He made no complaint but on questioning admitted that he had some headache. He required no medication.

produces headache and fever on its own account and is used for that purpose in protein shock therapy. The results were unsatisfactory,

and the clinical picture was ambiguous. I turned then to the use of solutions of dextrose. They are widely used in various conditions of low blood pressure, and their effect is fairly lasting. As the present routine procedure 4 ounces (124.4 Gm.) of 5 per cent dextrose in physiologic solution of sodium chloride is injected immediately following the operation. This introduces the dextrose solution at different times in the course of the anesthesia, but I have not been able to see that the result is thereby affected. Figures 8, 9 and 10 are charts for such cases.

This procedure was a surprising success and has very nearly stopped the study of headache. One patient admitted on questioning that he had a little headache. With that exception the material for study disappeared. My own interpretation of this result is that the anesthetic solution, even as nonirritating as I have been able to make it, still injures the delicate pia-arachnoid sufficiently to cause excessive absorption of cerebrospinal fluid and that this causes the headache which has been so troublesome. The slight hydremia which the dextrose solution induces is sufficient to check this excessive absorption until recovery or, probably, until compensation can take place, thereby reducing the disturbance in cerebrospinal hydrostatics to so low a level that headache does not appear.

SUMMARY

By using anesthetic solutions carefully made up to be as nearly nonirritating as possible, postanesthetic headache is reduced almost to the point of disappearance in patients in the recumbent position.

By means of the intravenous injection of 4 ounces of a 5 per cent solution of dextrose in physiologic solution of sodium chloride immediately after the operation headache is largely prevented even in ambulant patients.

NEUROFIBROMATOSIS

WITH REFERENCE TO SKELETAL CHANGES, COMPRESSION MYELITIS AND MALIGNANT DEGENERATION

ALEXANDER MILLER, M.D.
CINCINNATI

In its simpler form the typical pigmented spots on the skin and the multiple small subcutaneous nodules render neurofibromatosis easily recognizable. These characteristic changes, however, are not always present and at times are so overshadowed by other extensive and bizarre pathologic changes involving both the soft and the bony tissues that confusion in diagnosis may readily result. Numerous cases already reported, illustrating interesting changes of the skin, subcutaneous tissues, nervous system and skeleton, extend considerably the knowledge of the disease described by von Recklinghausen in 1882. To these a case of extensive involvement of the vertebral column leading to compression myelitis, with sarcomatous degeneration of more than one neurofibroma, seems worth adding.

REPORT OF A CASE

History.—H. M., a white boy aged 11 years, first came under observation in this department on March 12, 1924. The complaint was curvature of the spine, first noticed at 1 year of age, with a gradual increase in the deformity. In addition, a mass in the left inguinal region of several years' duration was noted. A history of the patient's birth, development and family revealed nothing unusual.

Physical examination revealed severe kyphosis (fig. 1) of the lower portion of the dorsal segment and the upper portion of the lumbar segment of the spine, with moderate scoliosis to the right and marked tilting of the pelvis. Over the posterior aspect of the right os ilium a typical elephantiasic skin flap about the size of a silver dollar was present. In the left inguinal region a similar formation was seen. The skin showed many pigmented spots characteristic of the disease. These had been present since birth.

Treatment for the kyphoscoliosis was instituted, and the patient was followed up for a period of five years, during which time little change in his condition was noted. He was not seen for several years, until he returned in July 1933 for a new brace. At this time he did not complain of unusual disability or discomfort and stated that he had been doing farm work. An increase in the spinal deformity, however, was noticeable.

The patient was not seen again until April 1934, when he was admitted to the hospital because of inability to walk. Six weeks previously some pain had been noticed in the region of the back. A week later the left leg felt numb and weak, and the next day the right leg became similarly involved. Three days later paralysis of both legs became complete, and this was followed the next day by

---

From the Department of Orthopedic Surgery, State University of Iowa, service of Dr. A. Steindler.

loss of control of the bladder and the bowels. Marked sensory disturbances were also noted. In addition, the patient stated that the mass in the region of the right flank had shown rapid growth during the past year.

Examination.—The patient, an emaciated man of 21 years, was of fairly normal height. The skin in general was dark, but on it, especially on the dorsal aspect of the trunk, were scattered many dark brown pigmented spots varying in size up to several inches in diameter. These were not elevated.

A striking feature was an enormous tumor on the right flank (fig. 2), about the size of a man's head, roughly spherical and covered with intact though

Figure 1

Figure 2

Fig. 1.—The patient as he appeared in March 1924. The kyphoscoliosis and cutaneous pigmentation are evident. The elephantiasic skin flap can be seen in the right gluteal region. The tumor on the right flank can be made out.

Fig. 2.—The patient in 1934. The tumor shows considerable growth, as does the elephantiasic skin flap below it.

atrophic and pigmented skin. The subcutaneous veins were distinctly enlarged. The lower half of the tumor was hard but showed smaller areas of varying consistency. The upper half was more elastic and in some areas gave the impression of fluctuation. Below the tumor an extremely large hyperplastic skin flap pre-

senting the characteristic appearance of elephantiasis covered the entire right gluteal region. Through this, several small tumors of the size and shape of a hen's egg were palpated. Their longitudinal axes ran parallel to the leg, and thick fibrous strands could be felt emerging from the poles.

A huge gibbous formation beginning at the eleventh dorsal vertebra and with its apex over the first lumbar vertebra was another prominent feature. There was some deviation of the spine to the right. The lumbar portion of the spine could not be made out distinctly, owing to the degree of kyphosis, but marked lordosis with rotation of the spinous processes to the right seemed to be present. The lower ribs were in contact with the iliac crest on the left side. Over the lower portion of the spine a circumscribed area of hypertrichosis was present in the midline.

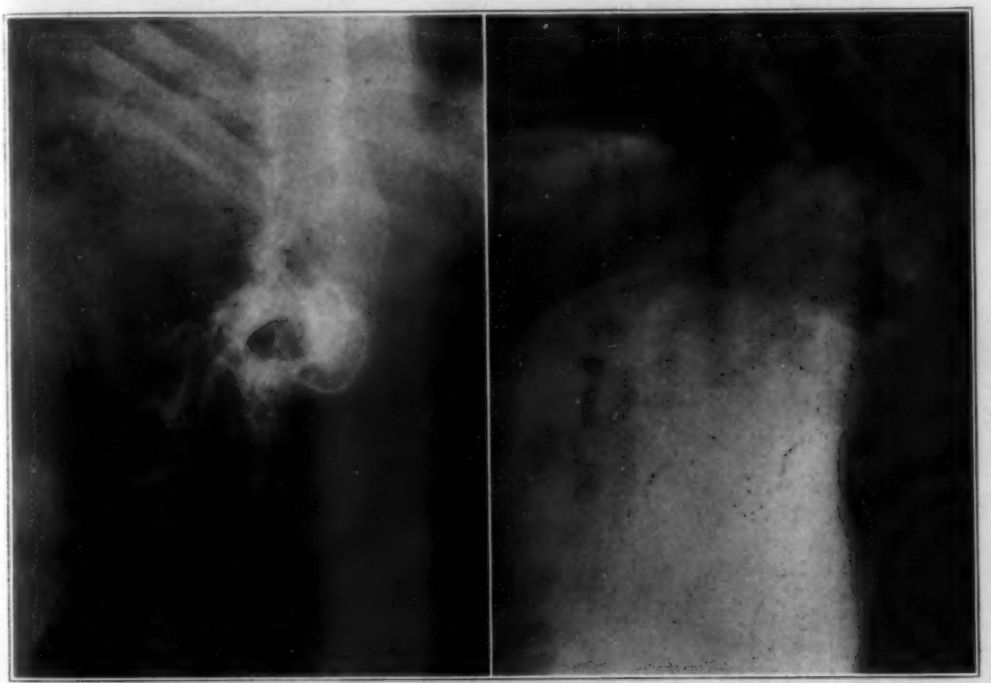


Fig. 3.—Anteroposterior and lateral views of the vertebral column, showing kyphoscoliosis, osteoporosis with compression of the vertebral bodies and deformity of the ribs.

The elephantiasic skin flap in the left inguinal region was firm and gave the impression of a hard fibroma. A small spindle-shaped tumor was visible on the outer side of the right wrist. Several small movable fibromas were palpated along the intercostal nerves. Palpation of the larger nerve trunks did not reveal other growths. The inguinal lymph nodes on the right side were enlarged.

The anteroposterior and lateral roentgenograms (fig. 3) showed a severe curvature involving the lower portion of the dorsal segment and the upper portion of the lumbar segment of the spine. Sharply kinked kyphosis with the apex at the first lumbar vertebra could be seen, to which was added a somewhat milder

scoliotic element. The vertebral bodies were porotic and collapsed and owing to the destruction of the intervertebral disks could not be differentiated from each other at the apex of the curve. In addition, deformity of the ribs was apparent.

Neurologic examination revealed a narrow, circumferential area of hyperesthesia around the trunk, below which hypesthesia and anesthesia involving all of both lower extremities was present. There was absence of tactile sensation and of the ability to distinguish between heat and cold. Motor paralysis of the bowels and bladder and of both lower extremities was complete. There was ataxia distally but not proximally. There was absence of the tendon reflexes.

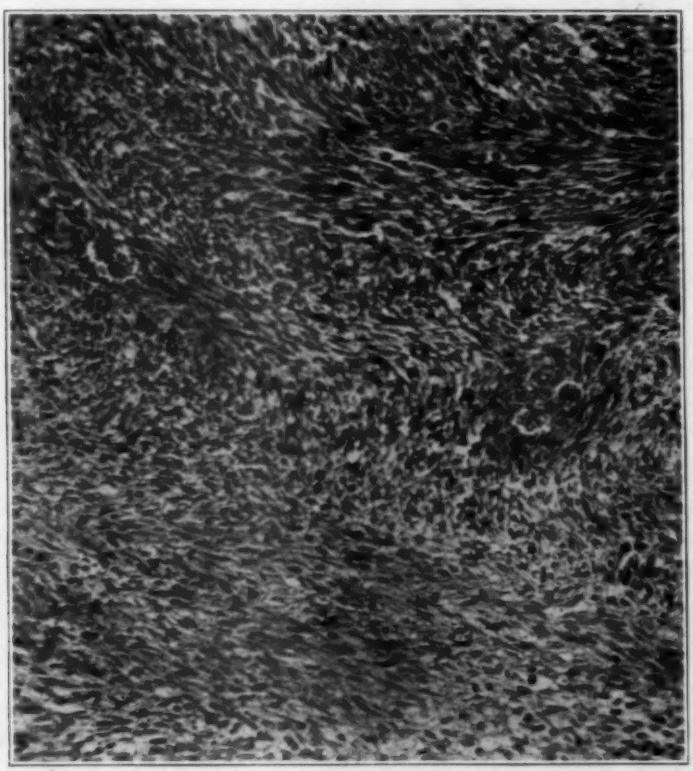


Fig. 4.—A medium power photomicrograph of the large tumor, showing spindle cell sarcoma with bundle formation.

The findings were typical for transverse myelitis beginning at the level of the first lumbar vertebra, the apex of the kyphoscoliotic curve.

Operation.—Because of the patient's poor general condition a laminectomy seemed inadvisable. However, an operation was performed to obtain a specimen for biopsy to determine whether the tumor was malignant. It was found that the large mass and the adjoining smaller ones could be easily enucleated, though there was invasion of the fascia lata of the gluteal and tensor muscles. Huge ramifying nerve trunks were found to enter and adhere to the tumor. In spite of the slight degree of trauma at operation, shock ensued, and the patient died in a few hours.

Biopsy.—The large tumor measured 10 by ½ by 5 inches (25 by 1.2 by 12 cm.), weighed 7½ pounds (3.4 Kg.) and was covered with a dense fibrous capsule except at the base, where the mass was torn and necrotic tumor tissue was present. The nerves entering the tumor were thickened and fusiform and on cross-section did not show definite nerve fibers. The tumor varied in consistency, and irregularly distributed in it were several cystlike formations containing a dark fluid. On section the tumor was seen to be edematous, complex, grayish white at the periphery and hemorrhagic in the central portion. Colliquation

Fig. 5.—A section through the cellular portion of the large tumor, showing a sharp line between the living and the necrotic tumor tissue. There is evidence of rapid growth, with failure of blood supply to keep pace.

and necrotic tissue could be distinguished, but a grossly malignant condition could not be definitely established.

On histologic examination the capsule was seen to be invaded in places by tumor cells and to show round cell perivascular infiltration. Some muscle was adherent to parts of the capsule, and in both these structures markedly thickened nerves were present. This enlargement of the nerves reached in some instances five times the normal size and was due to proliferation of the endoneurium

enclosed in a perineurium, which was also thickened. Normal nerve fibers were not evident. The vessels, though large, were of normal structure.

Beneath the capsule the living tissue was arranged in fibrous bundles (fig. 4). The structures were edematous and hyperemic. Under high power magnification the picture of a benign lesion was quickly dispelled. Among the more numerous fibrocytes were a number of smaller cells which were richer in chromatin and contained nuclei showing all the stages of mitotic division. The absence of a necrobiotic zone between the living and the dead tissue (fig. 5) and the presence of large areas which had become necrotic but still showed structural detail evidenced the rapidity of the growth of the tumor, with which the blood supply was unable to keep pace. Still further regression was seen in other areas, with

Fig. 6.—A section through the large tumor, showing colliquation resulting in the formation of a cyst with a good fibrous capsule.

complete destruction of the tumor tissue and the formation of cysts (fig. 6). In other places extensive hemorrhage by diapedesis and rhexis was evident.

The six smaller tumors removed were roughly of the size and shape of a hen's egg and showed thickened fibrous bands entering and leaving the poles. These tumors were hard and fibrillated, with areas of central necrosis which with a yellow sulfur-like stain suggested grossly mollusca fibrosa in different stages of catabolism. The fibrous bands consisted of nerves with marked proliferation of the interstitial and perineural connective tissue. In one of these tumors an angioma was present. All showed signs of early malignant degeneration (fig. 7). The yellow stain was imparted by the presence of huge cells which had foamy protoplasm and contained lipoid substance (fig. 8).

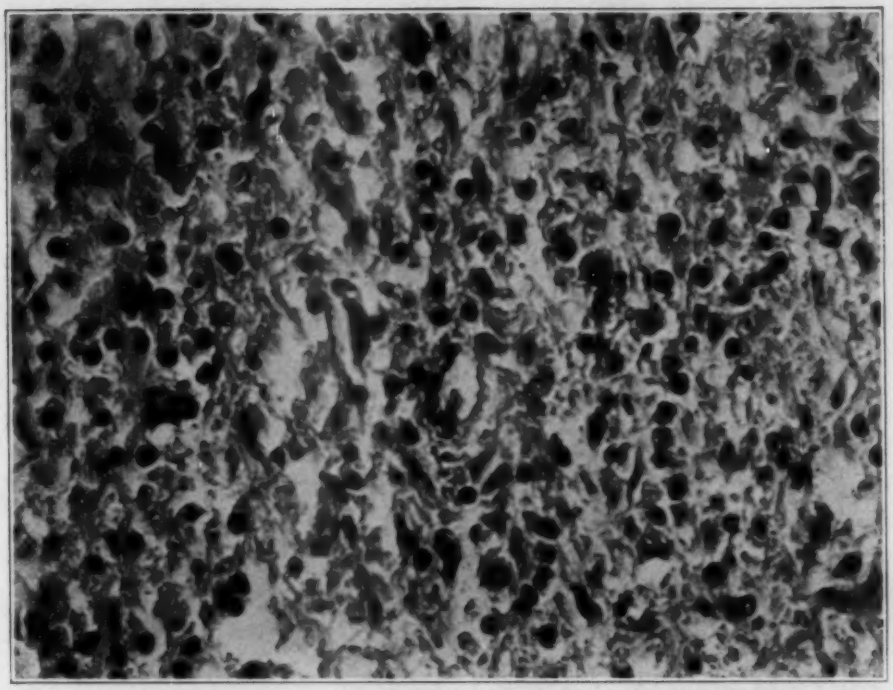


Fig. 7.—A high power photomicrograph of one of the smaller tumors, with anaplasia of cells indicating a beginning malignant process. Each of the six smaller growths showed this picture.

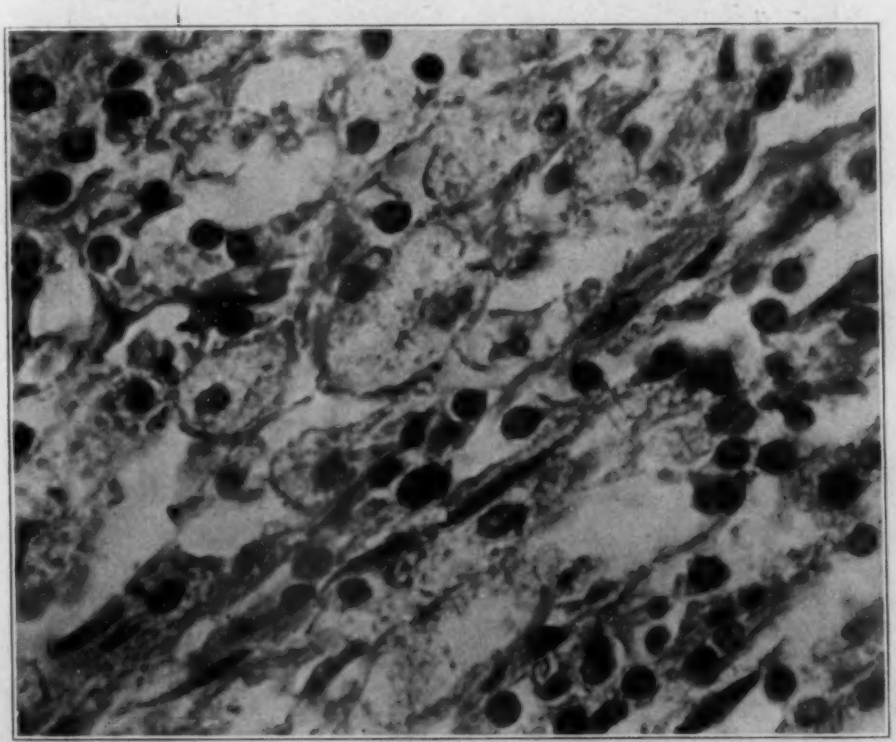


Fig. 8.—A high power photomicrograph through the yellow-staining portion of a small tumor, showing large, foamy cells.

The sections through the elephantiasic skin flap (fig. 9) showed marked fibrosis of the subcutaneous layer, with some attempt at the formation of bundles. The fibrous tissue was dense and almost hyaline. Where looser and more cellular tissue was present, peripheral nerves could be seen. These had undergone pathologic change and showed fibrous proliferation similar to that previously described. The sweat glands, in addition to being more numerous, were hyperplastic, owing

Fig. 9.—A section of the elephantiasic skin flap, showing fibrous proliferation of the subcutaneous layers, with thickening of peripheral nerve ends.

to an increase in connective tissue stroma similar in structure to that of the perineurium.

On histologic examination papillae in the skin were not seen. The epithelial layer showed little change except for a great amount of brownish granular pigment occupying the basal cell layer. The skin over the tumor was essentially normal except for areas which showed hyperpigmentation, chiefly in the striatum Malpighii but occasionally in the corium.

COMMENT

To the orthopedic surgeon the osseous changes in cases of neurofibromatosis are of particular interest. As the disease is considered a congenital disturbance of development, other congenital skeletal deformities may be coincidental. It is therefore not surprising that reports of spina bifida, anomalies of the ribs, defects of the fibula and exostoses of various bones are associated with Recklinghausen's disease. They, however, merely exist with, and do not form a characteristic part of, the skeletal picture in this condition. Of more importance are those alterations of bone which are incidental to the pathologic processes involved. They show certain typical clinical and roentgen features which help to complete the picture of the condition. Brooks and Lehman [1] described these changes, which can be grouped under the following four headings: (1) partial atrophy and arrest of growth, (2) local hypertrophy and hyperplasia, (3) local change of pressure due to the growth of adjacent tumors and (4) unexplained osteoporosis or malacia of the long bones and the vertebral column.

The skull and the bones of the face at times participate in the pathologic processes. Palpable defects of the cranium, over which plexiform neuromas or elephantiasis of the skin is present, were described by Kienböck and Rösler.[2] Thinning of the cranial vault, atrophy and aplasia of the mandible, maxilla and zygoma and involvement of the squamous portion of the temporal bone and of the mastoid process may be apparent on roentgen examination. Less often the changes are those of hyperplasia and hypertrophy, due, probably, to an irritative effect of the overlying neuromas. The combinations of atrophy and hypertrophy may lead to various asymmetrical distortions of the head and face.

In the long bones similar changes are occasionally found. When invasion of the bone by periosteal neurofibroma occurs, softening and deformity may result. When such changes are localized the picture of defects of the bone covered with a thin layer of periosteum can be demonstrated in the roentgenogram. These are the subperiosteal cysts described by Brooks and Lehman.[1] When stimulation is present, areas of hyperostosis are visible. The general outlines of the shafts of the long bones are irregular. With osteoporosis pathologic fractures may follow, some of which end with pseudarthrosis. Deformities, such as coxa vara, have been reported. Fliegel [3] inferred that neurofibromatosis

1. Brooks, B., and Lehman, E. P.: The Bone Changes in Recklinghausen's Neurofibromatosis, Surg., Gynec. & Obst. **38**:587 (May) 1924.

2. Kienböck, R., and Rösler, H.: Neurofibromatose: Eine Neurography, Fortschr. a. d. geb. d. Röntgenstrahlen, supp. 42, 1932.

3. Fliegel, O.: Knochenveränderungen bei Neurofibromatose, Deutsche Ztschr. f. Chir. **193**:359, 1925.

creates a disposition for the later appearance of osteitis fibrosa, a supposition which seems groundless. With periosteal hyperemia thickening of the bone may follow (Weber [4]).

In addition to the circumferential changes, abnormalities in the longitudinal growth of bone have occasionally been reported. When irritation is present, usually in areas where elephantiasis or plexiform neuromas are situated, an increase in the length of the bone may occur. This hyperplasia may reach the extent of gigantism, usually of a single extremity. Should decreased vascularization of the epiphyseal plate result, a shortened hypoplastic extremity may be expected. Such a hypoplastic extremity was present in one of Brooks and Lehman's cases as the result of destruction of the epiphyseal plate by a subperiosteal cyst.

These same processes of atrophy and hypertrophy and of hyperplasia and hypoplasia may involve the bones of the trunk, with resultant deformities and displacements, disturbances of growth and structural changes of the ribs and the pelvis. Here, also, may be found porosis, formation of cysts and areas showing bony defects.

Most frequent of all the changes in bone in cases of neurofibromatosis are those involving the spinal column. In about 43 per cent of the cases in which skeletal changes occur, the vertebral column is affected. The deformity is fairly characteristic and consists of kyphoscoliosis which differs in several important details from the usual types of scoliosis or kyphosis. The site of predilection is the lower portion of the dorsal segment of the spine. The deformity may first be noticed in early childhood. The kyphotic element strongly predominates, while the scoliosis is slight and presents a moderate degree of rotation. A typical feature is the presence of a sharp kink at the apex of the gibbus. Here a deep abrupt step may be seen and is palpable, so that the upper portion of the kyphotic part of the spine seems to overhang the lower part (fig. 1). The apex of the curve lies usually in the midline, and just below this point the rotation of the spinous bodies is most noticeable.

The cause of this collapse of the vertebral column, with at times a resultant dwarfism, has been variously explained. Gould [5] expressed the opinion that softening of the bone occurs which microscopically and macroscopically is indistinguishable from osteomalacia. In cases of neurofibromatosis, however, the loss of calcium salts is not so great as in those of the malacic disease. Other observers also have remarked on the similarity of the pathologic picture in the two conditions. Though

4. Weber, F. Parkes: Periosteal Neurofibromatosis with a Short Consideration of the Whole Subject of Neurofibromatosis, Quart. J. Med. **23**:151 (Jan.) 1930.

5. Gould, E. P.: Bone Changes Occurring in von Recklinghausen's Disease, Quart. J. Med. **11**:221 (April) 1918.

osteoporosis may result from invasion by periosteal neurofibroma, the process is too generalized to make this explanation entirely acceptable. It may be supposed that the high grade of porosis with the coexisting muscular weakness can account for the severe degree of deformity. That none of these assumptions make fully understandable the peculiar and rather constant pattern of the curvature must be admitted. The supposition that the vertebral bodies partake of a more generalized congenital mesoblastic disturbance is worth considering. In other words, this may be one manifestation of a generalized congenital disturbance of which the neurofibromatosis is the most constant feature. Gorlitzer's [6] case, in which there was rather severe deformity of the spine and the thoracic cage, would suggest such an explanation.

In addition to the kyphoscoliosis, all the osseous changes previously mentioned may be found in the vertebral bodies and arches. Erosion of these structures due to pressure effects from the growth of spinal neurofibroma, widening of the intervertebral foramina, local condensation and formation of cysts has been recorded.

Of unusual interest in the case reported here was the complete loss of motor and sensory control of the lower extremities, associated with loss of control of the bladder and the bowels. This is perhaps the most unusual complication of Recklinghausen's disease. Rarely has complete transverse myelitis with flaccid paralysis and anesthesia been reported in a case of this disease. One such case in the French literature [7] and one in the German, the case of Gorlitzer,[6] have been recorded. Several factors indicated that the lesion was one of true compression myelitis directly due to the spinal deformity. The absence of real pain, especially radicular, the rapid development of the neurologic manifestations and the extreme deformity at the apex of the kyphos, with correspondence of the site of involvement of the cord to this level, indicated strongly that the pathologic process within the spinal canal consisted of compression of the cord by the kinked vertebral column. Injury to the cord by hour-glass neurofibromas in the intervertebral foramina can be readily ruled out, as the development of the paralysis would have been much slower, and a unilateral lesion would have been expected first. Lesions of the cord of various degrees have been reported in cases of neurofibromatosis as the result of concomitant Pott's disease, spina bifida, or other anomalies of the vertebral column, but no such changes were found in my case.

---

6. Gorlitzer, Victor: Severe Neurofibromatosis Recklinghausen with Skeletal Deformities, Arch. f. Dermat. u. Syph. **159**:510, 1930.

7. Euzière; Lamarque, Viallofont and Longon-Turot: A Case of Recklinghausen's Disease with Kyphoscoliosis and Paraplegia, Arch. Soc. d. sc. méd. et biol. de Montpellier **10**:340 (July) 1929.

The observations of Jaroschy,[8] Grobelski[9] and Valentin and Putschar[10] on the occurrence of paraplegia in cases of severe curvature of the spine are applicable to the case reported here. That the myelitis in the reported cases was due to compression of the cord was proved by laminectomy by the first two investigators and at autopsy by Valentin and Putschar. The syndrome presents a typical picture. Except for the deformity of the spinal column, no cause is apparent. The paralysis develops without pain or fever and progresses rapidly to practically complete myelitis, involving also the bladder and the bowels. The paraplegia is as a rule spastic. The site of the lesion of the cord corresponds to the apex of the scoliotic curve, and on roentgen examination of the spinal cord a block can be demonstrated. The dorsal portion of the spine is almost always involved. The paralysis usually sets in in the latter half of the second decade of life, i. e., it corresponds to one of the periods of rapid growth. In 60 per cent of the twenty cases reviewed by Grobelski[9] the scoliosis was congenital. Originally, compression myelitis was thought to occur in patients with this type of scoliosis only, but later observations showed the occurrence of this lesion in patients with rachitic deformities of the spine. As to the mechanical origin of the lesion of the cord, compression by the kinked vertebral column, by bony or cartilaginous projections into the spinal canal and by torsion of the cord and dura were described by the authors just mentioned. Laminectomy is the treatment of choice and offers a good prognosis for recovery from the paralysis. The dura should be opened because of the torsion frequently present.

Except that the paralysis was of the flaccid type, the condition in the case reported here showed a distinct similarity to the features of compression myelitis associated with spinal curvature. The patterns of clinical history and age were identical and indicated a similar mechanical causation.

Pathologically the principal lesion in neurofibromatosis is proliferation of the endoneurium with hyperplastic changes in the perineurium. The process frequently proceeds to the point where all evidence of nerve fibers is lost. There may be localized thickening of the nerves palpable as small subcutaneous nodules or rather diffuse involvement resulting in the fusiform-shaped strands. The lesions may vary from the small, discrete seed-like tumor to the large, coarse communicating plexiform neuroma. The peripheral nerves are by far the most frequently and

8. Jaroschy, Wilhelm: Ueber Spätschädigungen des Rückenmarks (Kompressionsmyelitis) bei schweren Skoliosen, Beitr. z. klin. Chir. **142**:597, 1928.

9. Grobelski, M.: Kompressionslähmung des Rückenmarks bei Skolioser, Ztschr. f. orthop. Chir. **57**:220, 1932.

10. Valentin, B., and Putschar, W.: Clinical Pathology of Kyphoscoliosis with Damage to the Spinal Cord, Ztschr. f. orthop. Chir. **57**:245, 1932.

extensively involved, but neurofibromas of perhaps all the cranial, spinal and sympathetic nerves have been described. In their order of frequency, changes have been noticed in the ulnar, radial, orbital, gluteal, sciatic and crural nerves, according to Preiser and Davenport.[11] Pain is usually absent. If present it is neuralgic or neuritic. The nerves may be tender on pressure. Paralysis or atrophy rarely occurs.

The question of malignant degeneration of these tumors was fully studied by Hosoi.[12] He was able to collect reports of sixty-five cases from the literature. Of these, metastases occurred in fourteen cases, or 22 per cent, the lungs being most frequently invaded. In 72 per cent of these cases the condition developed after the third decade of life. Rarely did a malignant process occur primarily in more than one tumor nodule. The case reported here is therefore of additional interest in that malignant degeneration of various degrees was present in almost all the larger tumors studied (seven). Except for the advanced changes in the extremely large tumor, all the others were of similar appearance macroscopically and microscopically. Malignant degeneration had reached an equal stage in all. As no other structures were involved in the malignant process, one must suppose that on the basis of previously benign lesions sarcomatous degeneration took place simultaneously in several neurofibromas rather than that the changes were metastatic. The transition from a benign to a malignant process seems to have coincided with the period of rapid growth of the large tumor. This rapidity of growth was evident histologically from the sharp division between the living and the dead tissue without a necrobiotic zone and from the presence of structural outline where necrosis existed, indicating the inability of the blood supply to keep pace with the accelerated anaplasia. When the growth of a benign tumor undergoes unusual stimulation, the presence of malignant changes must be strongly suspected.

The typical coffee-colored spots and areas of elephantiasis have been too frequently described to necessitate further discussion. That these changes are dependent on a pathologic process in the subjacent nerves is a reasonable assumption in view of the widespread involvement of the peripheral nerves. Eller [13] reported several cases in which the typical cutaneous changes were present without any palpable neurofibromas having been found. However, as later observations revealed the

11. Preiser, Samuel A., and Davenport, Charles B.: Multiple Neurofibromatosis (von Recklinghausen's Disease) and Its Inheritance, Am. J. M. Sc. **156:** 507 (Oct.) 1918.

12. Hosoi, K.: Multiple Fibromatosis with Special Reference to Malignant Transformation, Arch. Surg. **22:**258 (Feb.) 1931.

13. Eller, J. J.: Incomplete Form of von Recklinghausen's Disease, Arch. Dermat. & Syph. **17:**648 (May) 1928.

development of such nodules, the nerves were probably already undergoing pathologic changes at the time of the original examination, though not yet clinically manifest. In the case reported here the cutaneous nerves underlying the hyperpigmented skin showed fibrosis microscopically, which was indiscernible grossly and which could not have been discovered on clinical examination.

Nothing new can be added concerning the etiology. The preponderance of changes involving the mesodermal tissue, especially the peripheral nerves, the subcutaneous tissue and even the sweat glands, lends further support to the hypothesis that neurofibromatosis is a manifestation of a congenital disturbance of the mesenchyme.

While there was no hereditary tendency in the case reported here, the presence of such a tendency was demonstrated particularly by Preiser and Davenport. They showed that there is also a family resemblance in the location of the tumors and in other expressions of the disease, such as the distribution of the cutaneous pigmentation.

In a certain number of cases mental deficiency appears. This feature was absent in the case reported here.

SUMMARY

A case of neurofibromatosis is reported in which there were, in addition to the typical nerve tumors cutaneous pigmentation and elephantiasis, more infrequent characteristics of kyphoscoliosis with compression myelitis and sarcomatous degeneration in more than one neurofibroma.

The changes of the bones in cases of neurofibromatosis are described, and their more important features are pointed out.

The relationship of spinal deformity to compression myelitis is discussed.

TRANSPLANTATION OF A TOE ACCORDING TO THE METHOD OF NICOLADONI

REPORT OF A CASE

PROF. M. I. KUSLIK
LENINGRAD, U. S. S. R.

The branch of surgery which is known by the name of "restorative surgery" and which aims at reestablishing lost functions and correcting cosmetic defects is one of the most fascinating. The possibility of restoring to the maimed their capacity for work and of freeing them from the mental depression caused by the realization of their infirmities is one of the noblest works a surgeon can perform: it brings him satisfaction and restores balance to the patient's life. Because of this, many of the most gifted surgeons have long since directed their efforts toward restorative surgery, which has achieved particularly great development within the last decade, largely in connection with the theoretical and practical study of plastic procedures. The healing on of parts of extremities which have been lost, in the accomplishment of which primary reimplantation and secondary grafting are distinguished as different methods, is of particular interest.

The healing on of completely severed finger-tips should be considered as the first experiment made within this particular field of surgery. Höpfner's experiments which resulted in partial adhesion of an amputated extremity in a dog served to show that it was possible to attempt reimplantation of even larger sections of the extremities. Jiame succeeded in reimplanting the forearm in man, and three years after the operation he had occasion to observe that the sensory, motor and vasomotor disturbances were considerably decreased. However, the success of similar operations demands a number of favorable conditions, and therefore the practical importance of reimplanting extremities is by no means great: Such cases as these will always be rare. On the other hand, secondary grafting which aims at compensating for the defects of such functionally important working levers as fingers seems to offer much greater possibilities.

The initiator of plastic surgical treatment of tendons, Nicoladoni, was also the first to make a new attempt in the second field of surgery. As early as 1891 he attempted to restore part of a thumb by means of a skin graft taken from the chest, with the intention of inserting

---

From the Clinic of Traumatology and Orthopedic Surgery of the Leningrad State Traumatological Institute (Director, Dr. F. I. Mashansky).

into this cutaneous sheath an osteoperiosteal wedge for support; he intended later to break the bone graft so as to adjust it at an angle most favorable for functional purposes. He was prevented from attaining his objective, because an amputation was performed by another surgeon. However, Nicoladoni's idea was adopted by other surgeons, and the operation has been repeatedly performed with good results (Nösske, Ritr and Neigesisser).

In 1897 Nicoladoni, not having lost his interest in these problems, suggested the brilliant idea of using toes to restore fingers, the operation being performed in two stages. A year later, to replace a maimed thumb he transplanted the second toe from the foot on the same side. In this case Nicoladoni met with partial failure; the distal phalanx of the transplanted toe became necrotic, and active motion in the first interphalangeal joint was not possible.

The clever idea of transplanting toes drew the attention of surgeons, and the operation has probably been performed often; however, so far only seventeen cases in which good results were attained have been reported. Fifteen of the operations were performed in other countries by: Nicoladoni (1898), Eisesberg (1900), Kraft (1904 and 1905), Krause (1906), Klemm (1917), Nörhammer (1915), Mühsam (1917), Payer (1917), Esser (1917), Riedel (1917), Oehlecker (1917 and 1919), Grégorie (1921) and Porzelt (1924). Two cases were reported in the Soviet Union by Professor Sviatukhin (1926) and Professor Wreden (1930). Consequently, my case is the third to be recorded in the Soviet Union. This comparatively small number of successful tansplantations is due to a certain complexity of the operation consisting in a number of technical details, each of which plays an important part if good results are to be achieved. Therefore a detailed description of every case in which success was attained is highly instructive in indicating how technical difficulties may be overcome. Of course, the description of failures with a detailed account of the causes is no less instructive, but, unfortunately, such cases are reported but rarely.

REPORT OF CASE

The patient, a girl aged 17, had received an injury of the left wrist while tobogganing. The accident was due to a breach of sporting rules; in the first place, the girl came down the toboggan-chute holding on to the edge of the sleigh, and in the second place, she and her companions started on their descent before the sleigh that had come down before them had been moved out of their way. The collision of the sleighs resulted in the girl's left index finger being completely wrenched away at the base of the middle phalanx and in a compound fracture of the middle phalanx of the middle finger.

The patient applied to the Institute in July 1933, complaining of an irregularly healed fracture of the middle phalanx of the middle finger and the deformity

of the index finger, which was the cause of constant depression. Within the same month the patient was subjected to the first stage of the operation performed according to Nicoladoni's method. Local anesthesia was employed. The distal surface of the stump of the finger was cut open by means of an arched incision, the tendon of the extensor muscle isolated, the surface of the phalanx exposed and the periosteum separated around it to a depth of about 0.5 cm. Furthermore, an incision forming a cutaneous flap was made at the base of the second toe of the left foot on the dorsal surface, both the dorsal interosseous arteries having been divided. The tendon of the extensor muscle of the toe was cut as far proximally as possible (fig. 1). One centimeter distal to the proposed level of division of the bone the periosteum was separated and turned back as a

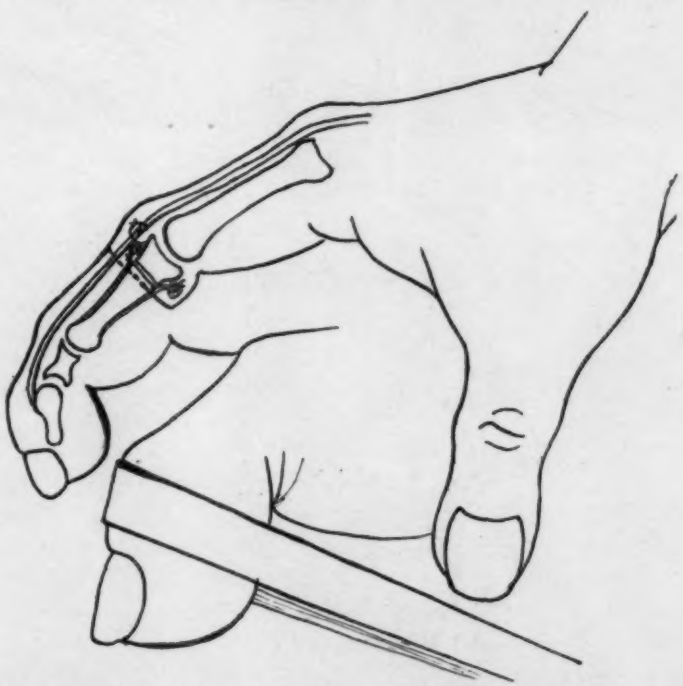


Fig. 1.—Drawing showing the first stage of transplantation of a toe on the index finger.

cuff, after which the bone was divided. The distal end of the phalanx of the toe was dislocated in the wound and adjusted exactly opposite the newly sectioned phalanx of the stump of the finger. The edges of the previously separated periosteum were fastened together by means of a circular suture of catgut with the object of strengthening the contact between the fragments of bone. The sutures binding together the ends of the tendons of the extensors were made somewhat proximal to those securing the periosteal edges, while suture of the skin was passed so as not to coincide with the level of that of the tendon. Finally, the patient having been placed in a sitting posture, a plaster of paris dressing was applied reaching to the upper edge of the shoulder blades (fig. 2).

In ten days an opening was cut in the plaster of paris bandage, and the regular pressing down of the plantar bridge was begun by inserting a tightly rolled ball

of cotton between the base of the bridge and the bandage. Only after a month had elapsed and the compression of the plantar bridge no longer affected circulation in the donor (the toe) did I attempt the second stage of the operation, i. e., the cutting away of the bridge and the suturing of the tendons of the flexor. Within the next two days the recipient (the finger) was kept warm by irradiation with an infra-red lamp through the bandage.

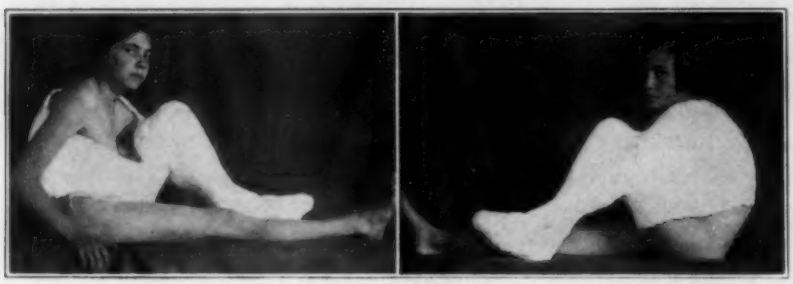


Fig. 2.—Photographs of the patient taken from each side, showing the adjustment of the cast during transplantation.

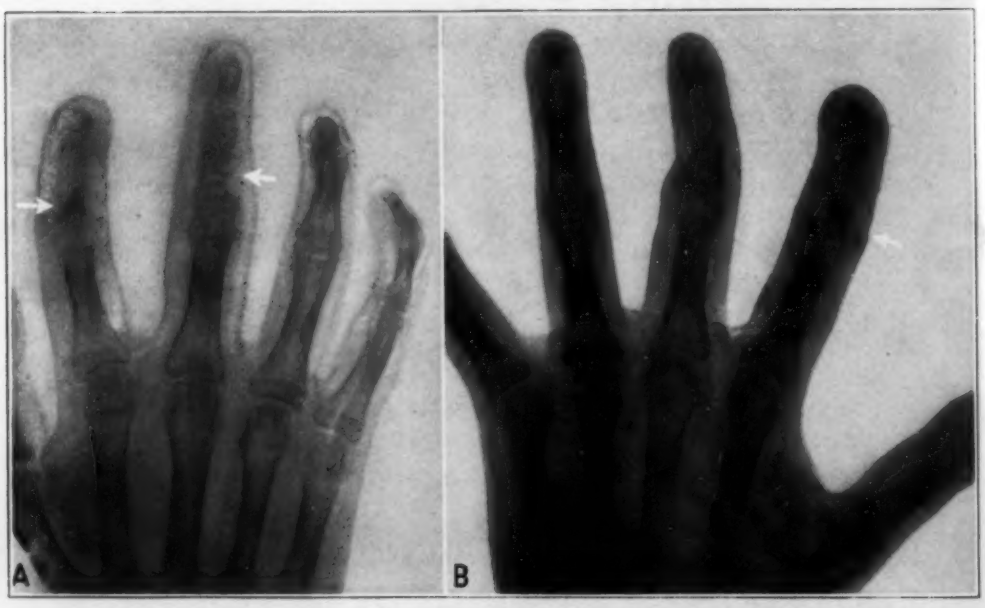


Fig. 3.—Roentgenograms showing the fracture of the middle phalanx of the middle finger (*A*) and healing after placing a wedge of bone between the phalangeal ends (*B*).

Seven days after the operation the sutures were removed, and the patient was treated by means of baths, massage, gymnastics and iontophoresis with potassium iodide in the outpatient department of the Institute.

Three months later the patient entered the Institute again for the correction of the badly healed fracture of the middle phalanx of the middle finger. Under

local anesthesia the bone was refractured. The distal end of the phalanx was then correctly adjusted, and a small wedge taken from the same phalanx was placed between the fragments so as to keep them in place (fig. 3).

Five months after the first operation the patient was presented before the Leningrad Society of Orthopedic Surgeons and the Pirogoff Surgical Society. Both the functional and the cosmetic results were found to be highly satisfactory (fig. 4). The line of grafting was manifest only by a narrow scar which was barely visible. The mental state of the patient was greatly improved.

Fig. 4.—Photographs showing good results of the operation on the index and middle fingers five months after the first operation.

In the case reported by Klemm sensation was not completely restored until the end of the second year after the operation. This may probably be accounted for by the fact that the scar along the line of grafting was somewhat thicker because healing occurred by second intention. My patient could use her finger to its full functional capacity, and it is of interest to note that the skin on the finger-tip had become somewhat callous; this was probably due to the fact that the skin of the toes is less adapted to work than that of the fingers. The strength

of the grasp and the pressure exerted by the thumb and the grafted index finger were no less than those of the other hand. The patient was much pleased with the results of the operation; the symptoms of mental depression had completely disappeared as soon as the defect of the finger was repaired, and the patient, who previous to the operation was always hiding her maimed hand, now readily submitted it to examination.

The foot with the amputated toe caused the patient no unpleasantness. Even in cases in which the thumb has to be replaced I believe it more rational to use the second instead of the great toe, both for cosmetic considerations and for preservation of the statico-dynamic function of the foot, in which the great toe is of the utmost importance. This statement is confirmed by the results in one of Oehlecker's cases in which the function of the foot was found to be disturbed subsequent to the grafting of the great toe.

On the ground of the aforementioned data the results in this case should be considered entirely successful, a fact which has been fully acknowledged at the meetings of the two surgical societies at which the patient was presented. By no means all of the eighteen reported cases of successful grafting of toes (the present case included) may be regarded as entirely satisfactory from the functional point of view. In ten of the eighteen cases the function of the recipient was barely satisfactory; in four cases (those of Klemm, Esser, Nörhammer and Oehlecker) the function was good, and in the four remaining cases (those of Riedel, Sviatukhin and Wreden and mine) the fingers completely fulfilled their normal function and proved to be genuine substitutes for the lost members. Thus, in performing the operation the surgeon has to cope with two tasks of increasing complexity: The first consists in insuring the healing of the grafted toe, and in my opinion the success of this part of the operation is chiefly determined by allowing the necessary time to elapse before the second stage, the separation of the bridge, is attempted; the second task is more complicated, for it consists in insuring active motor function in the finger, which makes it necessary to overcome certain technical difficulties.

In Prof. R. R. Wreden's clinic so far only two attempts to perform the operation have been made (those by Wreden, 1930, and me, 1933); both operations proved successful. I believe that the good results in my case should be attributed to the fact that a number of technical details were carefully worked out, and it is the chief object of this paper to present a full account of these details.

1. The pedicle should be made on the side of the sole, for the plantar arteries are better developed than the dorsal, and they therefore insure a better blood supply. Besides it saves the patient a great

deal of inconvenience to have the wrist placed on the dorsal surface of the foot with the fingers encircling it rather than fixed under the sole of the foot, the more so that in the latter case the hip and knee have to be brought into a position of still further flexion, which is especially hard to bear. Hence in performing the first stage of the operation I leave a plantar bridge with two plantar arteries.

2. It is of great importance as far as the functional success of the operation is concerned that the level of the osseous tendinous and cutaneous sutures should not coincide. When the case of the patient whom I presented was under discussion, Prof. J. J. Jenalidze suggested that, contrary to Bunnel's method, only the deep-lying flexor of the recipient should be sutured in the second stage of the operation, the superficial flexor being ignored, with a view to simplifying the technic.

3. To insure a better contact and fixation of the osseous fragments, suturing of the periosteal cuffs made previous to the osteotomy of the phalanx of the toe and freshening of the wound of the phalanx of the stump of the finger are advised.

With a view to obtaining a better contact between the ends of the phalanges Professor Sviatukhin advised in the first stage the resection of the superfluous central portion of the phalanx and the head of the corresponding metatarsal bone.

Fully appreciating the importance of the suggestion, I object to the resection of the head, for on this depends to a great extent the function of the foot.

4. Many of the patients find it extremely hard to endure the highly inconvenient position in which they are forced to remain for a considerable period of time. Their constant complaints have not infrequently led surgeons to attempt the second stage of the operation somewhat prematurely, a fact to which a number of failures may be attributed. With a view to alleviating these painful conditions I apply a plaster of paris bandage (fig. 2) which encases the upper and lower extremities on the side of the operation as well as the body, at the back reaching to the upper edge of the shoulder blades. In such a bandage the patient seems to sit leaning on the back of an armchair. The difficult problem of sleep is simply solved; the patient is put to bed lying on his side. A great deal may be gained by training the patient to maintain the necessary position prior to the operation.

5. Various authors have recommended different intervals between the first and second stages, the length of time varying considerably. For instance, Porzelt performed the second operation on the thirty-third day after the first, while Sviatukhin considered an interval of

seven days enough. Other authors have suggested intervals of varying duration within the range of the two just mentioned.

I believe that an interval can hardly be determined which will apply to all cases, as it depends on the establishment of nutrition in the pedicle. This is determined by the reaction shown in its circulation after compression of the pedicle. It is obvious that in different cases the interval will be of different length; it may depend on the patient's age, on his general condition, on the size of the graft, and other factors.

6. Within the first days after the operation I believe it highly advisable to keep the recipient warm by means of an electric bath, infra-red lamps, etc.

The strict observance of the technical suggestions just made will to a great extent insure the success of the operation. The patient's invalidism lasts from one and one-half to three months.

Taking into consideration the moral and economic effect of the procedure, I propose that it be more widely applied in the future, for the operation should no longer be regarded as a surgical experiment. It has become a well grounded and effective method of restoring ability to work and of overcoming cosmetic defects.

CONGENITAL ANOMALIES OF THE GALLBLADDER

A REVIEW OF ONE HUNDRED AND FORTY-EIGHT CASES, WITH REPORT OF A DOUBLE GALLBLADDER

ROBERT E. GROSS, M.D.
BOSTON

Congenital anomalies of the gallbladder are rare, but it is desirable for the roentgenologist and surgeon to be familiar with the malformations of this organ which they so commonly examine and operate on. Malformations of the bile ducts and their associated blood vessels have been admirably emphasized by Eisendrath, Haberland, Holmes, Howard and Wolbach, Kehr, Ladd, Mentzer and others, but such treatises have not included any comprehensive study of the gallbladder. It is my purpose in this presentation to collect from the literature the isolated reports relevant to the subject of anomalies of the gallbladder and to present this material in a unified manner. The review has been extensive but not exhaustive, for many articles have appeared in periodicals that were not available, and other examples have doubtless been overlooked. Numerous cases of the types under discussion were recorded before 1900, and while many of these were well described, only the more recent reports are employed. A study of the following 148 cases is adequate to illustrate the different types of abnormalities of the gallbladder. Histologic studies have usually been lacking, and the descriptions have been more concerned with gross findings. Notes on the vascular systems about these gallbladders are very poor, and in order that the course and distribution of the associated blood vessels may be better understood, future examinations and reports should include such data. Boyden's anatomic studies on the accessory gallbladder have been freely used in the preparation of the following sections.

ANOMALOUS FORMS OF THE GALLBLADDER

Double Gallbladder.—The term "double gallbladder" has been employed to describe 28 examples of reduplication of the vesica fellea in each of which there were two separate gallbladder cavities and two separate cystic ducts. The two cystic ducts may subsequently converge and form a joint cystic duct which enters the ductus choledochus (Y-shaped type), or the two cystic ducts may empty separately into the extrahepatic biliary system (ductular type). In the latter form the

From the departments of Pathology and Surgery of the Children's Hospital and the Harvard Medical School.

termination of the second or accessory cystic duct may be in the common duct or in one of the hepatic ducts. Of 22 cases in which the exact terminations of the cystic ducts were demonstrated, the "Y" type of duct was present in 7 (fig. 1*A*) and a "ductular" variety, or two completely separated ducts (fig. 1*B*), in 5.

An accessory gallbladder may be adjacent to the normal organ and thus lie in the normal fossa of the gallbladder (fig. 1*A*, *B* and *C*). When the two gallbladders lie next to one another they are often invested with a common peritoneal coat, and the true duplicate nature of the organ is therefore occasionally overlooked at the operating table. More

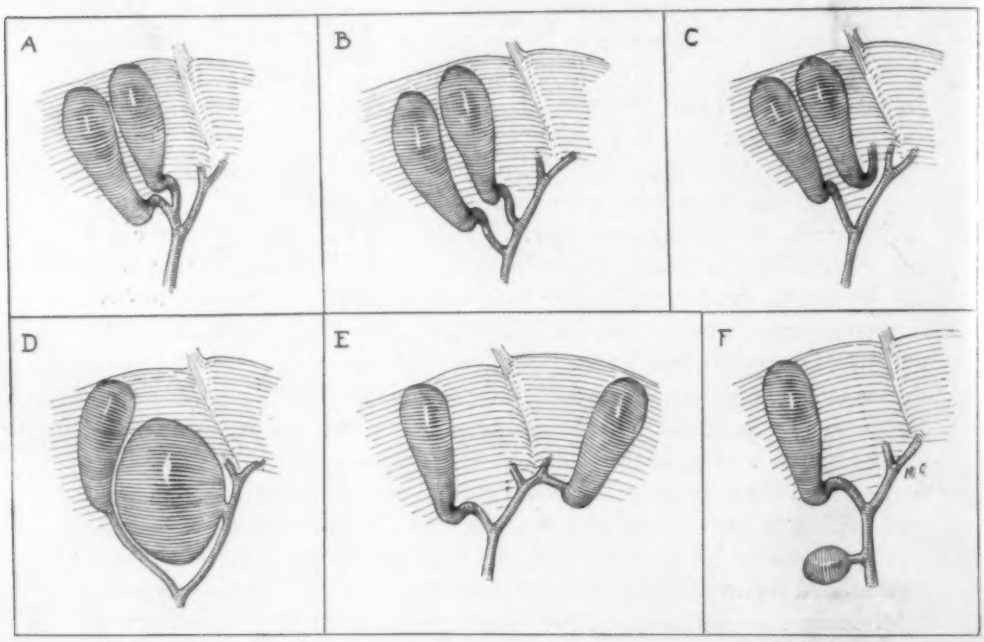


Fig. 1.—Types of double gallbladder, showing the positions of the accessory organs and the distribution of their ducts: *A* shows the gallbladder in the normal fossa with Y-shaped cystic ducts; *B*, in the normal fossa with two separate cystic ducts, and *C*, in the normal fossa with an accessory cystic duct directly entering the hepatic substance. *D* shows an accessory gallbladder partially embedded within the right lobe of the liver and communicating with the main hepatic duct; *E*, an accessory gallbladder under the left lobe of the liver and communicating with the left hepatic duct, and *F*, an accessory gallbladder in the gastrohepatic ligament and communicating with the common duct.

often there is a fissure between the two gallbladders, or else they are not covered by a common coat and the double structure is evident. The accessory gallbladder may lie in positions other than in the normal fossa. It may be situated under the left lobe of the liver and may communicate

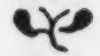
the left hepatic duct (fig. 1*E*). Another location is demonstrated in the case reported at the end of this section, in which the accessory organ was largely embedded within the right lobe of the liver and communicated with the main hepatic duct (fig. 1*D*). A fourth location is on the under-surface of the liver or within the gastro-hepatic ligament, the accessory organ having a communication with the common duct (fig. 1*F*).

The size of an accessory gallbladder is usually approximately the same as the normal structure, but occasionally it is only a half or two thirds of these dimensions. The anomalous gallbladders which arise from the common duct and lie along the gastrohepatic ligament are uniformly small and rudimentary structures which are from 1 to 2 cm. in overall length. The accessory gallbladder described here (fig. 1*D* and fig. 4) was much larger than the primary vesicle. In 23 cases for which such data are recorded, 17 duplicate vesicles occurred in females and 6 in males. Sixty-four per cent of these cases were discovered in the age groups between 30 and 60 years; the youngest person was 14 months of age, and the oldest, 69 years.

Boyden in 1926 collected and analyzed cases of accessory gallbladder in man and in animals. In the section on double gallbladder in human beings he listed 6 cases from the older literature which have not been included here. He also mentioned a specimen from the Hamann Museum for which few anatomic data were given. In a thorough study of animal material Boyden found 23 examples of double gallbladder in 2,600 cats, 47 cases in 2,555 calves and 3 cases in 2,560 lambs and sheep.

The embryologic explanation of the formation of these double vesicles varies somewhat with the form of the duplicate structure. A secondary gallbladder connecting with the cystic, hepatic or common duct probably arises from the small outpocketings which are so frequently seen on these ducts in embryonic life and which apparently represent a normal stage in the development of the extrahepatic biliary system. Such rudimentary and additional duct buds are common in human embryos and may spring from any one of the primary ducts, but they are most common at the junction of all three ducts (Keibel and Mall). Boyden found (in vertebrate embryos) a hepatic antrum at the junction of the cystic, hepatic and common ducts and showed that from this dilated portion of the ductal system there are numerous outgrowths and accessory vesicle formations. These supernumerary buds normally regress, but the persistence of one of them would doubtless result in formation of an accessory gallbladder. A second accessory gallbladder thus originating on the hepatic or common duct would be of the ductular variety, for it would connect with the biliary tree separately from the normal cystic duct. A second gallbladder arising

TABLE 1.—*Data in Cases of Double Gallbladder*

Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Roentgen Findings	Pathologic Observations	Type of Anomaly
1	Sherren	1911	F	25	Attacks of pain in right upper quadrant after meals for many years	Tenderness in area of gallbladder	.....	Symptoms from aberrant Brunner's glands in cystic duct; terminations of ducts?	
2	Schulz	1914	M	35	.....	.....	.....	Y-shaped cystic ducts, terminating in common duct; stones in both gallbladders	
3	Schachner	1916	F	52	"Gallstone colic for from 15 to 20 years"	.....	.....	Fissure between gallbladders; terminations of cystic ducts unknown; stones in gallbladders	
4	Haberer	1923	F	..	Severe gallstone colic	Gallbladder palpable	.....	Exits of cystic duct unknown; stones in both bladders	
5	Wischnewsky	1925	F	31	Fever, jaundice and weakness	Palpable, tense gallbladder	.....	Accessory gallbladder under left lobe of liver; terminations of cystic ducts (?)	
6	Braun	1926	..	..	.....	.....	.....	Cystic ducts with separate openings into biliary tree; one gallbladder half size	
7	Erdman	1926	..	..	.....	.....	.....	Y-shaped cystic ducts terminating in common duct	
8	Hardesty	1926	..	..	.....	.....	.....	2 cystic ducts, joined before entering common duct	
9	Lambert	1926	..	..	.....	.....	.....	Cystic ducts emptied separately into common duct	
10	Nichols	1926	M	51	Attacks of jaundice and pain in right upper quadrant and acholic stools for 10 yrs.	Tenderness in gallbladder area	2 rows of gallstones	Terminations of cystic ducts not stated; stones in both gallbladders	
11	Nichols	1926	..	..	.....	.....	.....	Exits of cystic ducts not determined	
12	Garafola	1927	F	37	Colicky pain in right upper quadrant, nausea and vomiting for 5 yrs.	Small mass in region of gallbladder	.....	One gallbladder half size of other; stones; Y-shaped cystic ducts (?)	
13	Priesel	1927	M	54	No symptoms from biliary tract (died of appendicitis)	.....	.....	Cystic artery behind both ducts and then in fissure between gallbladders	
14	Wakeley	1927	M	56	Attacks of abdominal pain, radiating to right shoulder and jaundice for 6 yrs.	Tenderness in right side of hypochondrium	Stones in 1 gallbladder	Y-shaped cystic duct; stones and inflammation of one gallbladder; other normal	

14	Wakeley.....	1927	M	56	Attacks of abdominal pain, radiating to right shoulder and jaundice for 6 yrs.	Tenderness in right side of hypochondrium	Stones in 1 gallbladder	Y-shaped cystic duct; stones and inflammation of one gallbladder; other normal	
15	Climan.....	1929	F	27	Epigastric pain following meals for 5 yrs.	Tenderness in right upper quadrant	Dye seen in 2 gallbladders	2 gallbladders functioned normally by cholecystography; no operation	
16	Judd, McIndoe and Marshall	....	..	..	.....	.....	.....	Terminations of cystic ducts not stated	
17	Wolfson.....	1929	F	19	Attacks of pain in right hypochondrium, with vomiting for 9 yrs.	Slight tenderness in right upper quadrant	Single group of stones	Accessory bladder 1.5 cm. long, with stones and unattached to liver	
18	Holderman.....	1930	F	30	Attacks of pain in right upper quadrant; chills and vomiting for 7 yrs.	Rigidity in right upper quadrant	Not visualized	Gallbladders adjacent to each other; openings of cystic ducts not stated	
19	Petrina.....	1930	F	..	Epigastric pain	.....	.....	2 separated cystic ducts	
20	Cave.....	1931	F	40	Vague abdominal symptoms, suggestive of pathologic condition in gallbladder	.....	2 shadows of dye seen	Inner vesicle functioned poorly; no operation	
21	Cave.....	1931	F	40	Symptoms suggestive of gallbladder disease	.....	2 shadows of dye seen	Outer vesicle functioned poorly; no operation	
22	Croudace.....	1931	F	52	Colicky pain of biliary type for 2 days; "dyspepsia" for several years	Marked tenderness in area of gallbladder	1 shadow seen	Accessory organ half size, with stone; accessory cystic duct led into liver	
23	Hayes.....	1931	M	35	Indefinite gastro-intestinal symptoms	.....	2 dye shadows seen	2 gallbladder shadows demonstrated by roentgenogram; no operation	
24	Strukow.....	1931	M	1	No symptoms referable to biliary tract (died of scarlet fever)	.....	.....	Incidental autopsy observations; 2 separate cystic ducts; opening into common duct	
25	Hermann, Hicks and Martin	1933	F	42	Epigastric pain, radiating to back; nausea, jaundice and pruritus	Icterus; tenderness in right side of hypochondrium	.....	Muscularis and mucosa in each gallbladder; stones in common duct and both gallbladders	
26	Kennon.....	1933	M	60	.....	....	.....	5 in. long gallbladder with stones; 3 in. gallbladder without stones; separate ducts	
27	Slaughter and Trout	1933	F	32	Attacks of pain in right upper quadrant; vomiting for 2 yrs.	Tenderness below right costal margin	Not visualized	Accessory gallbladder 5 cm. long, partly buried in liver; Y-shaped cystic duct	
28	Gross.....	1936	F	3	No symptoms referable to biliary tract (died of bronchopneumonia)	.....	No roentgenogram made	Accessory gallbladder 7 cm. in diameter, partly buried in liver, opening to hepatic duct	

from the normal cystic duct would produce a double structure with a Y-shaped cystic duct.

In contrast to those accessory gallbladders which arise from the extrahepatic ductular system there is a second form which has been designated the trabecular gallbladder (Boyden) because it arises from the hepatic trabeculae bordering the fossa vesicae felleae. Such a gallbladder always lies adjacent to the normal one, but its duct turns and courses directly into the substance of the liver. Such an anomalous structure probably represents an outpouching from the liver cell cords, and as such it develops a communication with the smaller bile capillaries. Boyden observed 4 of these accessory trabecular gallbladders in his series of calves and 1 in a sheep. The only example ever recorded in man is that published by Croudace (fig. 1*C*, table 1, case 22).

The reports of these cases do not give any characteristic symptoms or signs which might be of aid in diagnosing the presence of the congenital anomaly before operation or autopsy. When the accessory organ is the seat of inflammatory change or stone formation, the symptoms and signs are indistinguishable from those occurring with cholecystitis or cholelithiasis in a normally formed gallbladder. The mere presence of a second vesicle has not clearly given rise to symptoms in any case. Whether or not an accessory organ is more prone to be involved by disease processes is difficult to state, but the fact that most of these anomalies have been found at the operating table and only a few at postmortem examinations tends to show that the accessory structure is probably more likely to have pathologic changes than is a normally formed organ.

Roentgenographic examination has demonstrated a duplicate gallbladder in 5 instances. Climan in 1929 reported a case in which there was a second shadow which lay behind and below the first gallbladder shadow. Ten minutes after the ingestion of a fatty meal both shadows were reduced in size, and evidence of a distinct double gallbladder was more pronounced. Both vesicles showed good concentration of the dye and normal reduction in size after the fatty meal was taken. In Nichols' first case (1926) there were two distinct rows of stones in the region of the gallbladder which could not be approximated by manipulation of the gallbladder (fig. 2). In 1931 Hayes demonstrated two dye shadows indicative of a double organ (fig. 3). Cave (in 1931) recorded 2 additional examples of double gallbladders detected by cholecystography. In each of the last 2 cases, one gallbladder concentrated the dye and emptied normally while the contiguous gallbladder failed to empty properly.

A report of a case of double gallbladder follows:

G. B., aged 3 years and 3 months, entered the Children's Hospital in August 1932 because of a chronic infection of the respiratory tract of two and one-half

Fig. 2.—Roentgenogram of a double gallbladder showing two rows of stones in the two gallbladders. (Nichols' first case; reproduced from *Radiology* **6**:255, 1926.)

Fig. 3.—Roentgenogram of a double gallbladder, showing dye in two separate gallbladders. Other films showed the gallbladders spread apart. (Hayes' case; reproduced from *Radiology* **16**:66, 1931.)

months' duration. He died two days after entry to the hospital. At no time during the patient's life had there been any symptoms referable to the biliary tract.

Autopsy showed acute and chronic bronchopneumonia, bronchiectasis and fibrinopurulent and fibrous pleuritis. The observations of particular interest were limited to the liver and the biliary system. The liver weighed 500 Gm. and showed no evidence of cirrhosis or of biliary stasis. The under-portion of the liver was

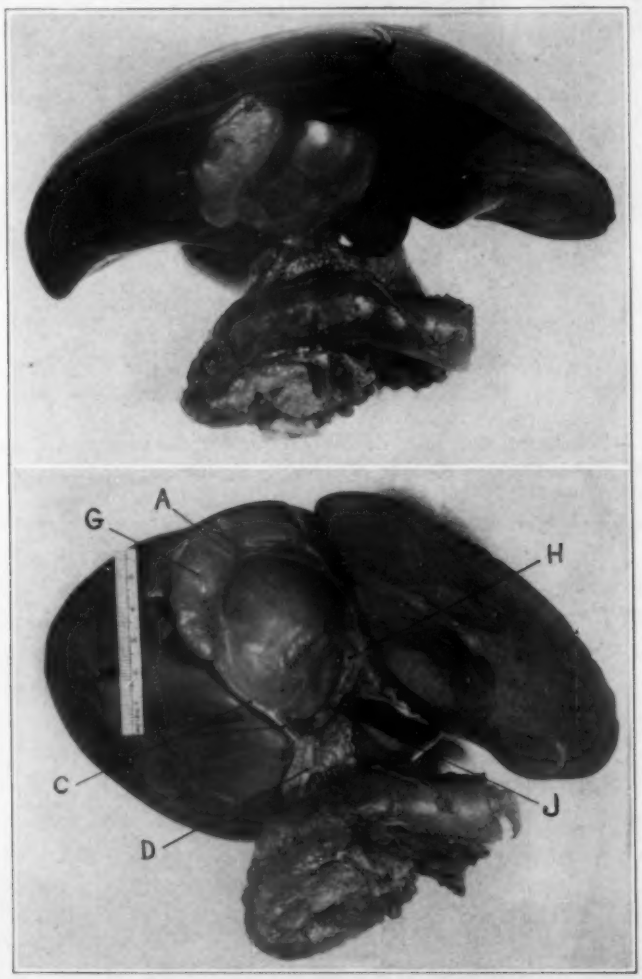


Fig. 4.—Photographs of the liver and double gallbladder in the case here reported. The accessory gallbladder is spherical and extends far up into the substance of the right lobe of the liver. *A* indicates the accessory gallbladder; *C*, the normal cystic duct; *D*, the common duct; *G*, the normal gallbladder; *H*, the hepatic duct, and *J*, the junction of the accessory gallbladder to the hepatic duct.

abnormal because of the presence of a large hemispherical cavity within the substance of the right lobe. The cavity was open below and extended up into the

liver, so that only a rim of the substance of the liver from 4 to 5 mm. thick remained at the dome of the right lobe. The cavity, which was about 7 cm. in diameter, lay above and to the left of the normal fossa of the gallbladder and was completely filled with a spherical, thin-walled accessory gallbladder which was 7 cm. in diameter (fig. 4). This bladder was filled with normal bile. Leading from the left lateral and the posterior portion of this saccular structure there was a small duct 3 mm. in diameter and from 4 to 5 mm. in length which communicated with the midportion of the main hepatic duct.

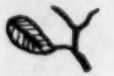
The primary gallbladder was normal in appearance and essentially normal in size for a patient of this age. It was 4.5 cm. long and 1.5 cm. in its greatest diameter. Its walls showed no evidence of inflammatory change. The cystic duct was 4.5 cm. in length. This elongated duct coursed around the right lateral aspect of the accessory gallbladder to enter normally at the junction of the hepatic and the common ducts. This primary gallbladder contained colorless mucinous fluid which at first could not be expressed down into the common duct and the duodenum. The presence of this white bile was apparently due to pressure on the primary cystic duct by the adjacent distended accessory gallbladder, for when the latter vesicle was deflated the primary cystic duct was found to be patent.

Microscopic examination of the wall of the accessory gallbladder showed it to be composed entirely of a dense band of hyalinized connective tissue. There was no smooth musculature. The inner layer lacked an epithelial lining. There were no mucosal glands. A few small masses of inspissated bile were found on some portions of the inner surface. There was no evidence of inflammatory reaction in the wall.

Bilobed Gallbladder.—This anomaly, which is so common in many of the higher vertebrates, is seldom encountered in man (table 2). Vesica fellea divisa, as the name implies, is a gallbladder with a bifid or partially divided structure. Included in this group are those specimens which have two cavities both of which are drained by a single cystic duct.

Two types of this malformation are seen. In the first of these the gallbladder appears normal to external inspection, but when opened it is found to have a longitudinal septum dividing the organ into two chambers. These two cavities communicate at their proximal ends and are supplied by one duct (fig. 5*A*). This septum is fibrous, but may contain a smooth musculature. Glandular elements have been described in the septal mucosa. The second form of bilobed gallbladder has a complete division of the fundic portion, with a fusion of these portions at the neck of the gallbladder. The cleft is shallow in some instances, but in other cases it almost completely divides the gallbladder into two parts. Externally such an organ has a V shape. The two cavities are thus completely separated at the distal ends but communicate with one another at the base and have a common exit through one cystic duct (fig. 5*B*). The two arms of such a gallbladder are usually of equal size, but occasionally one branch may be smaller than the other.

TABLE 2.—*Data in Cases of Bilobed Gallbladder*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Roentgen Findings	Pathologic Observations	Type of Anomaly
1	Deaver and Ashhurst.....	1914	F	55	No symptoms referable to biliary tract (death from apoplexy)	.....	.....	2 lobes of unequal size; calculus in larger one; other one normal	
2	Ryeroft.....	1923	F	42	Attacks of biliary colic with jaundice for 13 yrs.	Tenderness in right side of hypochondrium	.....	Gallbladder divided into 2 cavities by long septum; 1 calculus present	
3	Ullmann.....	1926	..	..	.....	.....	Seen by cholecystography	V-shaped structure; lobes separated at fundic portions	
4	Meyer.....	1926	F	55	Gallstone colic, fever and jaundice	.....	.....	2 separate but approximated lobes covered by a common peritoneal coat	
5	Phillips, Isaacs and Macdonald..	1931	M	48	(Recurrent pancreatitis and icterus)	Tenderness in left flank; jaundice	.....	V-shaped structure; marked inequality in size of lobes	
6	Phillips, Isaacs and Macdonald..	1931	F	56	Recurring attacks of biliary colic for 2 yrs.	.....	.....	Gallbladder cavity partitioned by central fibromuscular septum	

In the cat Boyden found that this was the most common anomaly of the gallbladder. He observed it 272 times, or in 10 per cent of all the animals examined. He also recorded 30 bilobed specimens in 2,555 calves and 27 in 2,560 lambs and sheep and 5 cleft gallbladders in 2,572 pigs.

The embryologic explanations of bifid V-shaped gallbladders has been clearly demonstrated in the cat (Boyden), and the same process possibly applies to man. In the cat the embryonic gallbladder frequently acquires a paired form by a subdivision of its primordium. When this structure is in the solid stage a splitting of it in a longitudinal direction changes this single bud into a partially divided organ. These paired buds maintain a connection with each other, but the fundic ends remain separated. The bilobed gallbladder in which the two cavities are separated only by a longitudinal septum probably represents an incomplete resolution of the solid stage through which the structure of the gallbladder

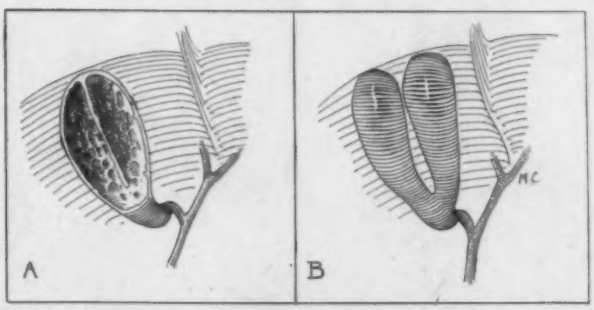


Fig. 5.—Forms of bilobed gallbladder having one cystic duct: *A*, a gallbladder with normal external contour but with two cavities separated by a septum; *B*, a gallbladder paired at the fundic portion and joined at the neck of the gallbladder.

normally passes. Instead of the entire cavity being again reestablished, a portion of the solid elements remains to form a fold or septum.

Diverticulum of the Gallbladder.—A diverticulum of the gallbladder may occur anywhere along the surface of the organ from the fundus to the neck (table 3). The diverticula vary in size from ¼ to 1½ inches (0.64 to 3.8 cm.) in diameter. Only a small number have been observed at operation, but they are occasionally recognized by cholecystography. Vastine recorded 2 well illustrated examples which were detected by roentgenographic examination, the first of which was confirmed by operation. He pointed out that a calculus in such a diverticulum can be detected by its separation from, but its constant relation to, the gallbladder shadow. The gallbladder shadow also appears to be slightly indented at the base of such a diverticulum.

TABLE 3.—*Data in Cases of Diverticulum of the Gallbladder*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Roentgen Findings	Pathologic Observations	Type of Anomaly
1	Kamerer	1912	F	48	Symptoms of a "well marked recurring cholecystitis"	.....	.....	Diverticulum 1½ in. in diameter in region of cystic duct	
2	Nadeau	1917	F	29	Pain in lower quadrant and epigastric pain for 3 mos.	Tenderness in right lower quadrant	.....	Large diverticulum projecting into liver and showing on anterior surface of liver	
3	Eisendrath	1918	..	..	.....	.....	.....	Diverticulum of body of gallbladder with stones; cysthepatic ducts passing into liver	
4	Abbott	1923	F	35	Attacks of pain in right side of hypochondrium and in back for 2 yrs.	Negative	.....	Diverticulum ⅛ in. long at neck of gallbladder	
5	Bársony	1927	F?	..	.....	.....	Seen in roentgenogram	Diverticulum of body of gallbladder; no operation	
6	Bársony	1927	..	..	.....	.....	Detected in roentgenogram	Hazelnut-sized diverticulum at fundus; no stones or inflammatory reaction	
7	Bársony and Friedrick	1928	F	23	Attacks of abdominal pain radiating to back with vomiting for 7 yrs.	Palpable gallbladder	Seen by cholecystography	Diverticulum of body of gallbladder	
8	Kremer	1932	..	38	.....	.....	Seen by cholecystography	Diverticulum of body of gallbladder	
9	Vastine	1934	F	38	Epigastric distress after meals	Epigastric tenderness	Stone in diverticulum	Diverticulum 1.3 cm. in diameter at fundus of gallbladder	

The origin of most diverticula of the gallbladder, particularly those occurring toward the neck, appears to be closely related to the development of the cysthepatic ducts. These small ducts, found in embryonic stages, pass from the gallbladder bud or from the upper portion of the cystic duct over to the substance of the liver. As the structures in the region assume their final size and relationships, these small cysthepatic ducts (when they persist) are found coursing between the neck of the bladder and the adjacent liver in the region of the fossa of the gallbladder. Such ducts are observed rarely in adult human beings, but Boyden has seen them in 25 calves and 2 pigs. An example in man is found in the case of Eisendrath (fig. 6*B*), in which there was persistence of the

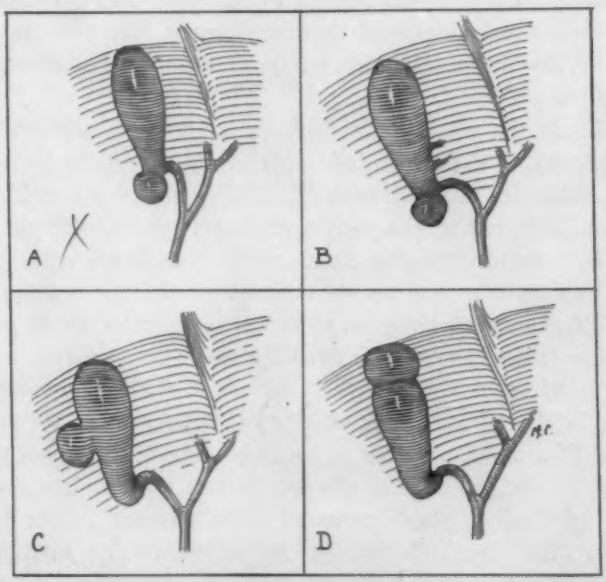


Fig. 6.—Forms of diverticula of the gallbladder: *A*, a diverticulum at the neck of the gallbladder; *B*, at the neck of the gallbladder, associated with persistent cysthepatic ducts which directly enter the gallbladder; *C*, of the body of the gallbladder, and *D*, at tip of the gallbladder.

cysthepatic ducts opening directly into the gallbladder. In this case a diverticulum was also present at the neck of the gallbladder. It is by persistence of these ducts, or from remnants thereof, that diverticula are believed to arise. While these ducts and early diverticula are thus present on that side of the gallbladder facing the liver, the normal rotation of the gallbladder may bring the diverticulum to lie on the exposed surface of the gallbladder (fig. 6*A*).

To explain the origin of a diverticulum on the fundus of a gallbladder is difficult. Those fundic forms which are described as having a trans-

verse incomplete septum pinching off a small cavity at the tip (fig. 6*D*) may be developed as an incomplete resolution of the solid stage through which the gallbladder passes in embryonic life. Thus a constricting band or incomplete diaphragm is left in the same manner as a congenital stenosis or atresia is developed in the small intestine.

Absence of the Gallbladder.—Total absence of the gallbladder is represented by the 38 cases listed in table 4. Only those cases are tabulated in which the hepatic and common ducts were present and functioning and in which the absence of the gallbladder was the only abnormality. There is a larger group of cases which are not included in this review. In these, in addition to absence of the gallbladder, there was atresia of all or some portion of the hepatic or common duct system. Approximately 200 cases of atresia of the extrahepatic bile passages have been recorded in the literature, and in about one sixth of these there was absence of the gallbladder.

Agenesis of the vesica fellea may occur without any other variation from the normal in the remaining extrabiliary system, as is demonstrated by the 38 examples listed in table 4. The fossa of the gallbladder was commonly absent, but occasionally a shallow fissure or dimple was visible on the under surface of the right lobe. Numerous authors observed that the quadrate lobe was poorly defined or entirely missing. Absence of the gallbladder was twice as common in females as in males. The ages of these patients when the anomaly was discovered, at operation or autopsy, varied from a few days to 65 years, with an average of about 35 years. Twelve of the abnormalities were observed in persons between the ages of 45 and 65. As far as could be determined from the reports, the absence of the gallbladder did not in any way impair the health or digestive functions of these persons. The absence of the gallbladder, per se, was rarely accompanied by any compensatory dilatation of the hepatic or common ducts, and many authors commented on the normal size of these structures. When the extrahepatic ducts were dilated the presence of gallstones raised the possibility that the increased size was due to obstruction from calculus rather than from absence of the gallbladder.

The absence of a gallbladder may be explained in two ways: The hepatic diverticulum arises from the foregut to form the liver, the extrahepatic ducts and the gallbladder. The gallbladder and cystic duct normally originate from this diverticulum as an outpocketing, but the failure of the latter development would cause complete absence of the gallbladder and cystic duct. A second way in which absence of the gallbladder might be explained is by the failure of the gallbladder bud to resolve from its solid embryologic stage. The hepatic, common and cystic ducts, as well as the gallbladder, are hollow structures in early embryonic life but later

TABLE 4.—*Data in Cases of Absence of the Gallbladder*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Pathologic Observations
1	Bubenhofer	1905	M	66	Died of cardiac failure	.....	No gallbladder or cystic duct; no gallbladder fissure
2	Niemack....	1908	F	60	Colicky pain in abdomen and right shoulder; clay stools; fever	Jaundice; fever	No gallbladder; stone in common duct
3	Stone.....	1908	F	54	Intermittent icterus and epigastric pain for 5 yrs.	Poorly nourished; anemic	No gallbladder; stones in hepatic and common ducts
4	Hinder.....	1909	M	60	Chronic discomfort after meals; attacks of apparent cholecystitis	.....	No gallbladder found; small dimple at edge of liver representing gallbladder fissure; chronic pancreatitis and hepatitis
5	Elben.....	1910	M	48	Nothing referable to biliary tract; died of pneumonia	.....	No gallbladder; no cystic duct; no gallbladder fissure
6	Elben.....	1910	F	20	Died of phthisis	.....	No gallbladder, cystic duct, gallbladder fossa or cystic artery; common duct of normal caliber
7	Hoffman and Jackson	1910	F	..	Attacks simulating cholecystitis	Elderly patient	No gallbladder; common duct greatly dilated
8	Leopold.....	1911	F	45	Attacks of colicky epigastric pain with icterus for 17 yrs.	Icterus; stools pale	No gallbladder; stones in hepatic and common ducts
9	Blakeway...	1912	M	..	No symptoms from biliary tract; died of imperforate anus	Newly born infant	No cystic duct or gallbladder; hepatic and common ducts not dilated; no quadrate lobe of liver
10	Goebel.....	1913	F	60	.....	.....	No gallbladder; stones in common duct
11	Ewers.....	1914	F	45	.....	.....	No gallbladder or cystic duct
12	Schulz.....	1914	F	60	Jaundice for 6 wks.	Jaundice; enlarged liver	No remnant of gallbladder; large stone in common duct
13	Attridge....	1918	..	..	Symptoms of obstruction of common duct	.....	No gallbladder; 2 stones in common duct
14	Müller.....	1920	F	28	Died of pneumonia	.....	No gallbladder, cystic duct or gallbladder fossa; common duct of normal size
15	Naegeli.....	1921	F	34	Gallstone colic, vomiting, icterus and clay stools, in attacks for 5 yrs.	Icterus; tenderness in right side of hypochondrium	No gallbladder; no gallbladder fossa; considerable dilatation of the common duct; stone in right hepatic duct
16	Naegeli.....	1921	F	65	Gallstone colic, radiating to shoulder; vomiting and icterus for 30 yrs.	Icterus; tenderness in right side of hypochondrium	No gallbladder; stones in common and hepatic ducts
17	Knox.....	1922	F	1	No symptoms from biliary tract; died of pneumonia and pleuritis	.....	No gallbladder or gallbladder fissure; common and hepatic ducts normal in size
18	Fuld.....	1924	M	20	Died of pulmonary tuberculosis	.....	No gallbladder or gallbladder fossa; no quadrate lobe
19	Giuliani.....	1924	F	35	.....	.....	No gallbladder; common duct contained stones and was dilated
20	Martin.....	1924	F	1	No symptoms from biliary tract; died of bronchopneumonia	.....	No gallbladder; slight groove present, marking right border of quadrate lobe
21	Jones.....	1926	F	3	Nothing referable to biliary tract; died after rectal operation	.....	No gallbladder

TABLE 4.—*Data in Cases of Absence of the Gallbladder—Continued*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Pathologic Observations
22	Nagel.....	1926	M	38	.....	.....	No gallbladder
23	Golob.....	1927	F	59	Gaseous eructations and feeling of pressure in epigastrium for 4 yrs.	Essentially negative	No gallbladder
24	Ishiyama...	1927	F	50	Yearly attacks of jaundice and pain in right side of hypochondrium for 29 yrs.	Icterus; acholic stools	No gallbladder; stones in common duct; common duct "enormously dilated" and thickened
25	Lintz.....	1927	F	48	Pain in right upper quadrant radiating to back for 5 yrs.	.....	Gallbladder absent; had a large Riedel's lobe on liver
26	Bower.....	1928	M	50	Severe pain right upper quadrant with jaundice	Tenderness and rigidity in right side of epigastrium	No gallbladder or cystic duct; no gallbladder fossa; absence of left lobe of liver; pancreatitis
27	Schmidt.....	1928	F	30	Colicky epigastric pain and loss of weight for 6 mos.	Liver enlarged and tender	No gallbladder or gallbladder fossa; gumma of liver
28	Brack.....	1929	M	22	.....	.....	No gallbladder
29	Meyer.....	1929	M	21	Colicky abdominal pain	Mild jaundice	No gallbladder
30	Machmull...	1930	M	57	Died of tuberculosis	.....	No cystic duct or gallbladder
31	Barnstorf...	1931	F	19	Died by drowning	.....	No gallbladder; common duct of normal size
32	Amadon....	1933	M	51	Chronic indigestion, pain in right upper quadrant and shoulder	Epigastric tenderness	No gallbladder or cystic duct; absence of quadrate lobe

have their lumens temporarily obliterated during the so-called solid phase through which all these structures pass. These lumens are all later reestablished, and the normal organs are formed, but an arrest in this obliterated stage produces atresia of any portion of the extrahepatic system (in the same manner in which intestinal atresia is derived). When there is atresia of the extrahepatic ducts or gallbladder there is usually a visible solid fibrous cord found representing ducts and gallbladder in infants who die of this condition. In contrast to these findings of a rudimentary cord in the fossa of the gallbladder in cases of associated atresia of the ducts, no such rudimentary structure was reported in any of the cases listed in table 4. The belief is therefore established that in the group of cases listed in this section the gallbladder is absent because of the failure of development of the gallbladder bud from the hepatic diverticulum.

Miscellaneous Malformations of the Gallbladder.—There are several forms of gallbladder which deserve only passing mention. An hour-glass gallbladder is frequently described, but from most of the reports it is not clear and is indeed impossible to tell whether it is the result of inflammatory change and subsequent cicatricial contraction. There are, however, examples in which this is undoubtedly a congenital malformation, particularly those occurring in younger children in whom there is

no evidence of an inflammatory process in the gallbladder. One is especially convinced of the congenital origin of such a form when it occurs with associated atresia or malformation of the bile ducts. Many cases of hour-glass gallbladder in the adult have been described after cholecystographic examinations, but such examples should not be accepted unless confirmed by operation, because adhesions or extrinsic masses pressing on the gallbladder may produce the picture of this deformity in the roentgenogram. A phrygian cap gallbladder has been frequently described as a congenital malformation, and many examples are recorded in which this has been found by roentgenographic study. This puckering over of the tip of the gallbladder is usually, if not always, an acquired characteristic. A rudimentary gallbladder is occasionally described. When they occur in elderly subjects it is impossible to tell how much is attributable to scar formation. There are, however, small rudimentary gallbladders found in infants and in children which are little more than a nubbin and which undoubtedly represent a congenital hypoplasia.

Of interest to the surgeon, in spite of their apparent rarity, are those variations in the bile ducts in which accessory ducts enter the gallbladder directly from the liver via the bed of the gallbladder. Four examples of this may be cited. Kehr described 2 cases, in each of which two large ducts directly entered the gallbladder on that side which lay against the liver. Odermott described an additional case, in which a large accessory duct thus entered the gallbladder. The same condition is probably represented in the case of Desgouttes and Ricard, in which an accessory duct coursed from the liver directly over to the neck of the gallbladder. These accessory ducts are persistent cysthepatic ducts which communicate between the gallbladder and the liver proper in embryonic stages. It is not impossible that many of these ducts have been overlooked and have been cut across in the course of operations on the biliary tract, and the severance of such ducts might well explain some of the leakage of bile which is occasionally encountered from the bed of the gallbladder.

ANOMALOUS POSITIONS OF THE GALLBLADDER

Abnormal sites of the gallbladder are rare, but at least four such locations should be considered because of the interest they have had for surgeons and the technical difficulties they have sometimes presented at operation (fig. 7).

Intrahepatic Gallbladder.—A gallbladder of this type lies wholly within the substance of the right lobe of the liver and is surrounded by hepatic tissue, so that at first sight the vesicle may be thought to be absent (fig. 7*A*). A small portion of the neck of the gallbladder may be visible, or a very small tip of the fundus may present as the gallbladder

protrudes through on the upper convex surface of the liver. A bulge of the anterior portion of the right lobe has aroused suspicion of an intrahepatic gallbladder, and in some cases incision of this mass has penetrated into the gallbladder cavity. Less striking examples of buried vesicles are known to surgeons and pathologists who are familiar with partially and deeply embedded vesicles which are only partly exposed on their under-surface. The tip of the gallbladder is occasionally hidden in the substance of the liver, while the remainder of the organ is essentially free. In some instances the gallbladder is mostly exposed, but a bridge of hepatic tissue enfolds and completely encircles the body. The intrahepatic gallbladder is the extreme and more rare form of these

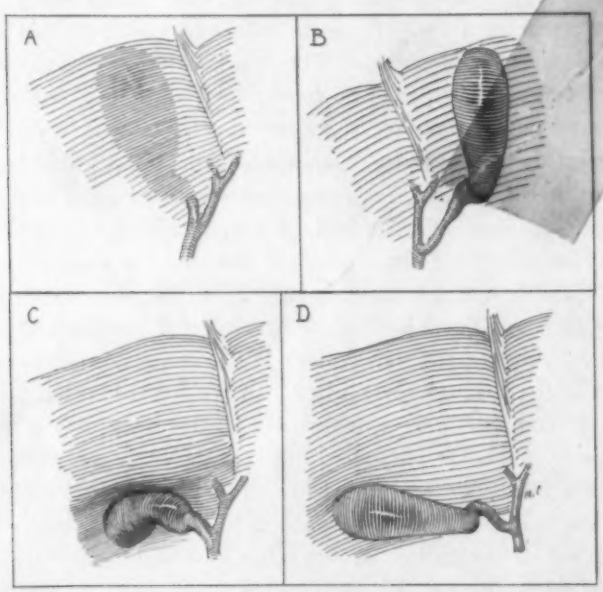


Fig. 7.—Abnormal positions of the gallbladder: *A*, within the substance of the liver; *B*, under the left lobe of the liver; *C*, on the posterior part of the inferior surface of the right hepatic lobe, and *D*, horizontally, within the transverse fissure.

variations. In the case of Walter and Neiman the distance between the surface of the liver and the buried gallbladder was from 3 to 4 cm., and no cystic duct was seen outside of the liver. The experience of Stetten demonstrated that an intrahepatic gallbladder containing stones is better treated by incision, removal of the stones and drainage than by removal of the gallbladder with its attending shock and hemorrhage.

Gallbladder on the Left Side.—A gallbladder of this type has been described at least 7 times. In each instance the vesicle was situated

on the under-surface of the left lobe of the liver (fig. 7*B*). It was usually of essentially normal size, but in the first case of Kehr it was small. The cystic duct of the gallbladder entered the left hepatic duct in 1 case, entered normally at the junction of the hepatic and the common ducts in 3 cases and had an unrecorded exit in the other 3 instances. In all cases the gallbladder lay to the left of the round and falciform ligaments, and where the latter ligament was very broad the gallbladder was hidden from the operator's view through an ordinary incision in the right upper quadrant. In none of the 7 cases was there any gallbladder in the fossa under the right hepatic lobe.

A gallbladder can be developed to lie under the left lobe of the liver in one of two ways: First, the ectopic gallbladder which had a normal exit for its cystic duct very likely arose as a normal embryologic bud from the hepatic diverticulum, but instead of assuming its normal position under the right lobe it migrated to a position under the left lobe. It would therefore retain its normally implanted and attached cystic duct. With the development of the peritoneum the gallbladder would be fixed in its position to the left of the falciform ligament. Such an origin seems highly probable in cases such as those described by Walton and by Harris, in each of which the cystic duct arose from the junction of the hepatic and the common duct, was directed forward and to the right toward the normal fossa of the gallbladder and then took a sharp hairpin turn toward the left and passed behind the falciform ligament to communicate with the gallbladder on the left side. A second way in which a gallbladder on the left side may be explained is as an independent development of a second gallbladder directly from the left hepatic duct, accompanied by a failure of development of the normal structure on the right side. This appears to have been the process in the first example recorded by Kehr.

Retrodisplacement of the Gallbladder.—This abnormality is exemplified by 5 reports. The gallbladder is not found in its normal position but has a cystic duct arising in the usual fashion from the extrahepatic system. The gallbladder does not normally course forward and downward on the under-surface of the liver but instead leads upward and backward to gain the inferior and posterior portion of the liver (fig. 7*C*). In Mayo Robson's case it was attached to the under-surface of the liver, but in Baldwin's case it passed downward and backward and lay just to the right of the vertebral column. In Ferguson's first case the gallbladder was not visible, but it could be felt with a palpating hand as it lay retroperitoneally, extending along the vena cava in an upward, backward and outward direction. In Ferguson's second case the gallbladder could not be seen, but it could be felt on the posterior edge of the under-surface of the liver and behind the peritoneum. The diffi-

culties encountered in draining or removing stones from such an organ are obvious. Such a position of the gallbladder is probably attained by migration of the normally formed structure and an anchoring in this backward portion by the developing peritoneum.

Transverse Position of the Gallbladder.—This condition has been described twice. In these 2 instances the gallbladder lay somewhat farther back than its normal position and was turned to assume a horizontal position within the transverse fissure (fig. 7*D*). In Gay's case the gallbladder was barely one-third the normal size and lay in the extreme right of the transverse fissure; it was almost completely surrounded by the substance of the liver. Deaver and Ashhurst also described a gallbladder which was deeply placed and "horizontally transverse." Such a site for a gallbladder is again presumably dependent on an abnormal rotation or migration of the definitive organ.

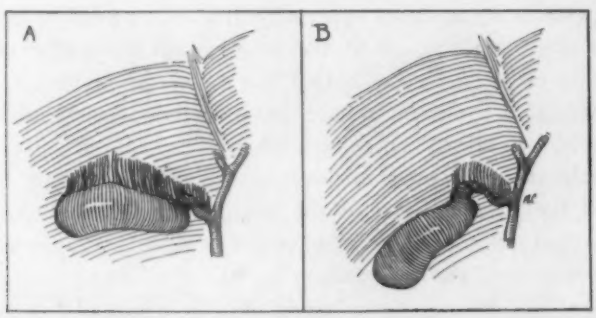


Fig. 8.—Floating gallbladders with a "mesentery:" *A*, mesentery supporting the gallbladder and cystic duct; *B*, mesentery supporting only the cystic duct, allowing the gallbladder to hang free.

Floating Gallbladder.—Torsion of the Gallbladder: As has been previously seen, the gallbladder may assume positions with varying degrees of partial or complete embedment within the liver. Of similar interest, but of greater importance from the standpoint of frequency of occurrence and liability to produce clinical symptoms, is the study of gallbladders which hang from the liver on a sort of "mesentery." The peritoneal coat surrounding such a gallbladder continues as two approximated leaves to form a fold or mesentery to support the gallbladder from the under-surface of the liver. This fold is of variable length and may allow the gallbladder to hang for as much as from 2 to 3 cm. from the inferior hepatic surface. The fold may run the entire length of the gallbladder and cystic duct (fig. 8*A*), or it may support only the cystic duct and allow the gallbladder to hang free and movable (fig. 8*B*). Graded forms and combinations of this fold have been described, the

classification being based on the length of the fold and the amount of the gallbladder which is supported thereby. In different series of investigations the number of gallbladders possessing this supporting membrane has been listed as high as 4 to 5 per cent of dissecting room specimens.

It is not difficult to foresee the clinical significance of this anomaly, for an organ which is so freely movable is apt to become twisted, and infarction will supervene from impairment of the blood supply in the small pedicle. Cases of this sort have been frequently listed in the literature, and 50 examples are listed in table 5 for summary and study. All cases with a gallbladder "mesentery" are too numerous for listing; hence only those have been included in which there was torsion of the gallbladder and operation or autopsy has been performed because of this condition.

The clinical symptoms of torsion of the gallbladder are strikingly similar from case to case and strongly suggest the proper diagnosis. This torsion is most frequently seen in women in the upper age groups. Of these 50 cases, 84 per cent were in women and 16 per cent were in men. The average age of the patients was 59 years. Of the 50 patients, 8 per cent were in the age group between 50 and 60 years; 16 per cent in the age group between 60 and 70, and 34 per cent in the age group between 70 and 80; 2 patients were over 80.

The patients are almost always thin, ptotic persons. The symptoms related to torsion of the gallbladder have an abrupt, rapid onset and continue with extreme severity. Intense and constant pain in the right upper quadrant ushers in the attack and is present during the entire length of the illness. Pain is rarely described as being transmitted to the back or to the right shoulder. Nausea and vomiting are always marked. Collapse is frequently encountered. The temperature, if at all elevated, is rarely over 100 F., but commonly the pulse rate is rapid and ranges between 100 and 120 or even as high as 150. The severity of the symptoms naturally predicates symptoms of short duration, and the history is rarely of more than from twenty-four to forty-eight hours' standing. Oddly enough, there is usually only one attack from this condition. One would expect, with a freely movable organ, that there might be minor episodes of abdominal pain, vomiting, etc., from temporary twists of the vesicle, but such does not seem to be the case. In only 5 cases was there any previous complaint which might have been attributable to torsion of the gallbladder and spontaneous relief. In the majority of instances, then, the patient has only one attack of torsion of the organ.

Physical examination shows an elderly, thin patient, usually a woman. Exhaustion is common. Tenderness in the right upper quadrant and spasm are the rule. The gallbladder is often palpable (54 per cent of

TABLE 5.—*Data in Cases of Abnormally Placed Gallbladders*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Signs	Pathologic Observations
					Intrahepatic Gallbladders		
1	Dévé.....	1903	..	..	.....	.....	Gallbladder within substance of right lobe of liver
2	Lemon.....	1905	..	..	.....	.....	Gallbladder entirely embedded within substance of right lobe of liver
3	Loughran.....	1905	M	..	No symtoms referable to biliary tract; killed in mine explosion	.....	Normal sized gallbladder, almost entirely embedded in hepatic substance; cystic duct partially embedded in hepatic tissue
4	Walter and Neiman	1911	F	27	Attacks of pain in right upper quadrant and nausea; occasional vomiting	Temp., 101.2 F.	Liver incised and gallbladder found; 2 stones removed; distance between surface of liver and gallbladder 3 to 4 cm.; cystic duct in liver
5	Stetten.....	1933	M	45	Attacks of epigastric pain, fever and vomiting for 12 yrs.; jaundice once	Tenderness in right subcostal area	Gallbladder entirely surrounded by hepatic substance, except for a small area on its superior aspect; stones in gallbladder
					Left-Sided Gallbladders		
1	Kehr.....	1902	..	..	.....	.....	Gallbladder under left hepatic lobe and situated to left of ligamentum teres; cystic duct entered left hepatic duct
2	Dévé.....	1903	..	..	.....	.....	Gallbladder to left of ligamentum teres; opening of cystic duct not recorded
3	Walton.....	1902	F	..	Nothing referable to biliary tract; died after prolonged labor	Newly born infant	Cystic duct started up toward right, then turned to left, to enter gallbladder which was to left of ligamentum falciforme
4	Kehr.....	1913	F	35	Colicky pain in right upper quadrant for 6 yrs. radiating to back and loss of weight	.....	Gallbladder under left lobe of liver and to left of ligamentum teres; stone in neck of gallbladder, opening of cystic duct (?)
5	Virenque.....	1923	..	..	.....	.....	Gallbladder under left hepatic lobe
6	Harris.....	1926	M	1 Mo.	No symptoms referable to biliary tract; died of bronchopneumonia	.....	Cystic duct started to right, then turned to left to join gallbladder; bladder under falciform ligament with fundus to left of midline
7	Rolleston and McNee	1929	..	..	.....	.....	Gallbladder under left lobe of liver
					Retrodisplacement of Gallbladder		
1	Ferguson.....	1901	M	45	Gallstone colic, jaundice and fever for 4 yrs.	.....	Gallbladder not visible; stones palpable in gallbladder; gallbladder retroperitoneal, extending from vena cava upward, backward and outward
2	Ferguson.....	1901	M	75	Gallstone colic with colitis for 1 yr.	.....	Gallbladder not seen; felt on posterior edge of inferior surface of liver, behind peritoneum; stones
3	Mayo-Robson..	....	..	..	.....	.....	Gallbladder coursed downward and backward on posterior inferior surface of right hepatic lobe
4	Baldwin.....	....	..	..	.....	.....	Practically no left lobe to liver; gallbladder passed downward and backward, lying just to right of vertebral column
5	Hofmann.....	1925	M	23	Colicky pain in region of gallbladder for 4 yrs.	.....	Gallbladder ran backward and downward toward kidney, lying in bed on under-surface of liver; cholecystitis and stones in gallbladder

TABLE 5.—*Data in Cases of Abnormally Placed Gallbladders—Continued*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Signs	Pathologic Observations
					Transversely Placed Gallbladders		
1	Gay.....	1902	M	27	No symptoms referable to biliary tract; died of nephritis and endocardtiis	.....	Gallbladder (⅓ normal size) in extreme right of transverse fissure of liver and almost surrounded by hepatic tissue
2	Deaver and Ashhurst	1914	M	38	Pain in area of gallbladder with jaundice for 4 yrs.	.....	Gallbladder deeply placed in horizontally transverse position; calculi in gallbladder, cystic duct and in common duct

the cases). The twisted gallbladder is usually felt just below the edge of the liver, but because of its mobility it is sometimes felt in other parts of the abdomen, as in the right lower quadrant or in the umbilical region. A temperature of over 100 F. is rare, unless the gallbladder has ruptured and peritonitis has set in. When the white blood cell count has been listed, it has ranged up to 18,000 and 20,000. In cases uncomplicated by peritonitis, the white blood cell count and the pulse rate are much higher than might be expected with the associated temperature.

At operation the gallbladder is found to be twisted on its pedicle in an arc varying from 90 to 1,080 degrees. Torsion of from 180 to 360 degrees is most often described. With the twisting of the pedicle the cystic artery and vein are occluded or compressed, and infarction of the gallbladder results. The wall of the gallbladder is therefore swollen, edematous, hemorrhagic and necrotic. Hemorrhagic fluid is occasionally found within the peritoneal cavity, and fibrinous exudate is usually deposited on the surrounding structures. The nature of the congenital anomaly makes surgical relief easily and quickly possible, for no dissection of the bed of the gallbladder is necessary. Rapid placement of clamps and ligatures on the vesicle stump and vessels is all that is required before the gallbladder can be cut from its pedicle.

The operative results in this condition have been very promising. Of the 47 patients operated on, 70 per cent recovered and 15 per cent died; the outcome in the remaining cases is not stated. These results are especially noteworthy in view of the age of these persons.

The occurrence of torsion of the gallbladder in elderly persons is peculiar in view of the fact that the gallbladder has been freely movable during the entire life of these patients. The onset of torsion is probably delayed until later decades, when supporting fat has largely disappeared from the surrounding structures and when atrophy of the tissues allows for greater ptosis of the viscera, including the floating gallbladder. These two factors tend to increase the mobility of such a gallbladder and enhance the possibility of torsion.

TABLE 6.—*Data in Cases of Bladder with Mesentery and Torsion of Bladder*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Pathologic Observation	Treatment and Result
1	Mayer	1908	F	54	Abdominal pain 2 days before hospitalization; no vomiting	Abdomen rigid; pulse 150; collapse; mass in right side of abdomen	Gallbladder infarcted; rotated twice on cystic duct; stones in gallbladder	Cholecystectomy; death
2	Mühsam	1908	F	61	Pain in right side radiating to shoulder for 4 days; vomiting for 1 day	Abdomen spastic; tender on right; mass near umbilicus; white blood cells, 20,000	Gallbladder enlarged, red-black and turned 360 degrees clockwise	Cholecystectomy; recovery
3	Nehrkorn	1908	F	74	Severe pain in right side and back for 8 days; general condition poor	Tender mass in area of gallbladder larger than a fist	Gangrenous gallbladder hung free on pedicle, on which it was twisted	Cholecystectomy
4	Lett	1909	F	72	Sudden pain in right side of hypochondrium for 3 days; vomiting 4 or 5 times	Right side of hypochondrium very rigid and tender; mass felt; temp., 101 F.	Gallbladder black; twisted twice; mesentery supported only cystic duct	Cholecystectomy; death
5	Fischer	1910	F	70	Repeated vomiting and pain in right side for 2 days; general ill health	Abdomen tense and spastic; mass in right side of abdomen; looked very sick	Gallbladder black-red and tense; twisted 360 degrees in clockwise direction	Cholecystectomy
6	Kubig	1912	M	73	.....	.....	Gallbladder twisted 270 degrees; discolored; local peritonitis	No operation; death
7	Cramp	1915	M	..	Abdominal pain, nausea and vomiting	Rigidity in right upper quadrant; epigastric mass; temp., 99 F.	Gallbladder twisted one and a half times counterclockwise; gangrenous	Cholecystectomy
8	Schachner	1916	F	45	Digestive disturbances; vague pain in upper part of abdomen	Movable mass felt in right upper quadrant	Mesentery supported gallbladder as well as cystic duct	Cholecystectomy
9	Reichel	1919	F	71	Sudden illness, abdominal pain and vomiting for 3 days	Right side of abdomen tender and resistant; pulse rate, 88	Fluid in abdomen; gallbladder gangrenous; twisted 360 degrees	Cholecystectomy; recovery
10	Krabbel	1920	F	73	Epigastric pain, nausea and gaseous eructations for 4 days	Abdominal distention; tender fist-sized mass felt under liver	Gallbladder twisted 360 degrees; discolored and gangrenous; contained stones	Cholecystectomy; recovery
11	Krabbel	1920	F	64	Severe abdominal pain and vomiting for 1 day; previous gallstone colic	Abdomen distended; gallbladder enlarged, palpable and tender	Gallbladder discolored; turned 360 degrees on pedicle	Cholecystectomy; recovery
12	Krabbel	1920	F	78	Abdominal pain and vomiting for 4 days (occasional abdominal pain for 2 yrs.)	Abdominal distention; tender, resistance under liver	Gallbladder twisted 180 degrees and contained stones; discolored	Cholecystectomy; recovery
13	Hansen	1921	F	79	Acute onset of abdominal pain; nausea and vomiting for 1 day	Tense, tender mass in area of gallbladder	Fluid in abdomen; gangrenous gallbladder, twisted 360 degrees	Cholecystectomy; recovery
14	Irwin	1921	F	34	Sudden severe epigastric pain and pain in back with frequent vomiting for 1 day	Abdomen rigid; tenderness on right side of abdomen; temp., 99 F.	Gallbladder darkly colored, with loose adhesions; turned 360 degrees on pedicle	Cholecystectomy; recovery
15	Strauss	1921	F	42	Sudden severe abdominal pain and vomiting ("stomach pain" for 1 yr.)	Mass felt in region of pylorus	Gallbladder 16 cm. long and 7 cm. in diameter, infarcted, with several stones	Cholecystectomy; recovery
16	Frankau	1922	F	62	Colicky pain in upper part of abdomen and incessant vomiting for 1 day	Rigidity and tenderness, especially in right upper quadrant; temp., 97.6 F.	Gallbladder black, edematous and distended; twisted 360 degrees	Cholecystectomy; recovery

17	Ormos	1922	F	71	"Died with symptoms of hemorrhagic colitis"	.....	Greatly enlarged, necrotic gallbladder; turned 450 degrees; peritonitis	No operation; death
18	Jonas	1923	F	67	Sudden onset of generalized abdominal pain, vomiting and collapse	Sausage-shaped mass felt below liver	Gallbladder enlarged and black; twisted 2½ turns; stones in gallbladder	Cholecystectomy; recovery
19	Dubs	1924	F	..	Sudden onset of abdominal pain	Elderly patient; pear-shaped mass in area of gallbladder	Enormous, movable gallbladder, with narrow mesentery; twisted on duct	Cholecystectomy; recovery
20	Daux	1925	F	11	Sudden onset of generalized abdominal pain and frequent vomiting	Abdominal spasm; temp., 99 F; pulse rate, 112	Gallbladder distended and black; peritonitis; twisted 180 degrees clockwise	Exploration only; death
21	Fifield	1925	F	54	Sudden onset of severe pain right upper quadrant and vomiting for 2 days	Tenderness, rigidity and mass in right upper quadrant	Gallbladder black, distended and very mobile; rotated once	Cholecystectomy; recovery
22	Fischer	1925	F	44	Attacks of pain in back and right shoulder with vomiting for 3 yrs.	Tenderness over area of gallbladder	"Mesentery" 4.5 cm. long and 2 cm. high; gallbladder twisted 90 degrees	Cholecystectomy; recovery
23	Fischer	1925	F	29	Attacks of pain in epigastrium, back and right shoulder	Marked tenderness in right side of epigastrium	Gallbladder hung 3 cm. below liver; twisted 90 degrees	Cholecystectomy; recovery
24	Lutaud	1925	F	56	Sudden pain in right side of hypochondrium	Tender, tense, mass felt on right side of abdomen	Gallbladder had double twist	Cholecystectomy; recovery
25	Sutter	1925	F	62	Symptoms of incarcerated umbilical hernia for 1 day	Tense mass in umbilical hernia	Gallbladder on mesentery, twisted 180 degrees	Cholecystectomy; recovery
26	Sutter	1925	F	72	Recurring abdominal pain and vomiting for 6 days	Movable fist-sized tumor on right side of abdomen	Tensely filled, pedunculated gallbladder, twisted 360 degrees	Cholecystectomy; recovery
27	Sutter	1925	M	71	Pain in right upper quadrant for years, especially when lying on left side	Slight tenderness over area of gallbladder	Gallbladder filled with stones; freely movable; twisted 90 degrees	Cholecystectomy; recovery
28	Feldmann	1926	F	46	Sudden epigastric pain and vomiting	Marked spasm and tenderness in right abdomen; temp., 99 F.; white blood cells, 18,000	Gallbladder size of "infant's head," and infarcted; twisted 180 degrees	Cholecystectomy; recovery
29	Feldmann	1926	F	74	Acute onset of epigastric pain and vomiting	Spasm and tenderness in right epigastrium; obese; temp., 99.5 F.	Gallbladder dark red; infarcted; twisted 180 degrees	Cholecystectomy; recovery
30	Grunert	1926	F	66	Attack of abdominal pain 3 days previously (gallstone colic for 15 yrs.)	Fist-sized tender mass in right upper quadrant; icterus	Gallbladder hemorrhagic; twisted 180 degrees; stones in gallbladder and common duct	Cholecystectomy; recovery
31	Grunert	1926	F	67	Illness with severe unceasing pain in right epigastrium and vomiting for 2 days	Fist-sized, tender mass in right side of abdomen; temp., 99.8 F.; pulse rate, 110	Fluid in abdomen; bladder gangrenous; twisted 360 degrees; stones	Cholecystectomy; death
32	Huddy	1926	F	71	Sudden onset, right-sided pain for 2 days (bilious attacks for 17 yrs.)	Tenderness, rigidity and mass about umbilicus; temp., normal	Gallbladder twisted once, anticlockwise	Cholecystectomy
33	Jenckel	1926	M	..	Pain in right side of hypochondrium for 4 days; relief if patient remained quiet	Elderly patient; tenderness in right upper quadrant	Gallbladder infarcted and gangrenous	Cholecystectomy; death
34	Jenckel	1926	M	76	Sudden pain in right upper quadrant and collapse; less when patient quiet	Tenderness in region of gallbladder	Gallbladder gangrenous	Cholecystectomy; death

TABLE 6.—*Data in Cases of Bladder with Mesentery and Torsion of Bladder—Continued*

No. of Case	Author	Year	Sex	Age, Yrs.	Symptoms	Physical Findings	Pathologic Observation	Treatment and Result
35	Marinacci	1926	..	..	Sudden onset of symptoms	Mass felt in region of gallbladder	Gallbladder distended, black, necrotic; twisted on cystic duct	Cholecystectomy; recovery
36	Busch	1927	F	75	Epigastric, intermittent pain for 3 days	Spasm and tenderness in upper part of abdomen; mass felt near umbilicus	Gallbladder enlarged, hemorrhagic and twisted	Cholecystectomy; recovery
37	Carless and Wakeley	1927	..	..	Symptoms for 24 hrs.	.....	Gallbladder twisted on its hepatic attachment; gangrenous	Cholecystectomy
38	Esau	1927	F	48	2 attacks of sudden illness, right epigastric pain, vomiting and collapse	Spasm and elongated mass in right lower quadrant	Fluid in abdomen; gallbladder edematous and thickened; twisted 90 degrees	Cholecystectomy; recovery
39	Esau	1927	F	..	Severe, 24 hr. attack of pain and vomiting	.....	Gallbladder hung free from surface of liver; mucosa necrotic	Cholecystectomy
40	Holden	1927	M	49	Abdominal distention, nausea and subsequent epigastric pain for 1 day	Right subcostal rigidity and tenderness; white cell count, 11,400	Gallbladder black and gangrenous; twisted on its cystic duct 1½ times	Cholecystectomy; recovery
41	Shipley	1927	F	65	Sudden onset of continued epigastric pain, radiating to back for 2 days	Looked ill; entire abdomen rigid; temp., 100 F.; pulse rate, 100	Serosanguineous abdominal fluid; gallbladder smooth and black; torsion 360 degrees	Cholecystectomy; recovery
42	Therstappen	1928	F	60	Abdominal pain, especially on right side, for 2 days	Emaciated; tender mass under right subcostal margin	Gallbladder turned three complete revolutions; solitary stone in gallbladder	Cholecystectomy; recovery
43	Meade	1929	F	36	Recurring pain on right side of abdomen for 1 day	Rigidity and tenderness of abdomen, especially over appendix	Fluid in abdomen; gallbladder distended and gangrenous; rotated three times	Cholecystectomy; recovery
44	Costantini	1930	..	42	Diffuse abdominal pain, especially in area of gallbladder, with vomiting	Spasm and pear-shaped mass, in right side of hypochondrium; pulse rate, 120	Green-black, edematous gallbladder; twisted on pedicle	.....
45	Gwynne	1930	F	77	Sudden severe pain in right subcostal area, with nausea and vomiting	Tender, pyriform mass in right side of hypochondrium; moved with respirations	Gallbladder reddish purple; twisted clockwise 180 degrees	Cholecystectomy; recovery
46	Kettner	1930	F	55	Sharp sudden pain in right side of epigastrium associated with vomiting	Mass in right upper quadrant; pulse rate, 100	Gallbladder hemorrhagic; gallbladder contained stones; twisted 180 degrees	Cholecystectomy
47	Wakeley and Hunter	1930	..	..	Severe colic	.....	No infarction; colic relieved by removal of bladder with mesentery	Cholecystectomy; recovery
48	Wakeley and Hunter	1930	..	..	Severe colic	.....	No gangrene of gallbladder; symptoms relieved by removal of gallbladder	Cholecystectomy; recovery
49	Wakeley and Hunter	1930	..	..	Symptoms for 24 hrs.	.....	Gallbladder twisted on attachment and gangrenous	Cholecystectomy
50	Murray	1933	M	83	Unabating pain in right upper quadrant, with frequent vomiting, for 2 days	Tenderness and rigidity in right side of hypochondrium; white cell count, 22,000	Bloody fluid in abdomen; gangrenous gallbladder; twisted 540 degrees	Cholecystectomy; death

SUMMARY

One hundred forty-seven cases of congenital anomalies of the gallbladder are selected from the literature for review. A case of double gallbladder is reported.

The presence of a double gallbladder has been recorded 28 times in man. The accessory bladder may be contiguous to the normal organ, under the left lobe of the liver, partially within the substance of the liver or rarely along the gastrohepatic ligament.

A bilobed gallbladder has been occasionally described in man. This may have the form of a single organ, divided by an internal central septum, or more often has a V shape (with the two cavities joined only at their junction with the cystic duct).

A diverticulum of the gallbladder may occur along the free surface of the organ from the neck to the fundus, and in 1 instance a diverticulum was described on the hepatic side of the gallbladder.

Thirty-eight cases of absence of the gallbladder are listed in which there was no other anomaly of the liver or biliary system. (Approximately 35 other cases of absence of the gallbladder have been recorded in which there was associated congenital atresia of the extrahepatic ducts.)

A normally formed gallbladder has been found in the following anomalous positions: within the substance of the liver, under the left lobe of the liver, posteriorly under the inferior aspect of the right hepatic lobe and horizontally in the transverse fissure of the liver.

A "floating gallbladder," because of its supension by a "mesentery," is likely to become twisted and infarcted. The resultant gangrene of the gallbladder is productive of severe clinical symptoms and necessitates immediate operation. Surgical removal of the organ leads to recovery in most instances if operation is performed before peritonitis supervenes.

BIBLIOGRAPHY

Abbott, G. K.: Diverticulitis of the Gall Bladder, Surg., Gynec. & Obst. **36**:466, 1923.

Amadon, P. D.: Agenesis of the Gall Bladder Associated with Pancreatitis, Am. J. Surg. **19**:263, 1933.

Attridge, J. A., in discussion on Eisendrath, D. N.: Anomalies of the Bile Ducts and Blood Vessels, J. A. M. A. **71**:867 (Sept. 14) 1918.

Baldwin J. F. (case of abnormally located gallbladder), cited by Mayo-Robson, A. W.: Operations upon the Pancreas and Bile Passages, in Burghard, F. F.: A System of Operative Surgery, New York, Oxford University Press, 1909, vol. 3, pt. 1.

Barnstorf, F.: Zur Agenesie der Gallenblase, Frankfurt. Ztschr. f. Path. **42**:304, 1931.

Bársony, T.: Seltenere Gallenblasenbefunde, Fortschr. a. d. Geb. d. Röntgenstrahlen **36**:759, 1927.

Das Röntgenbild eines Gallenblasendivertikels, München. med. Wchnschr. **74**: 546, 1927.

——and von Friedrich: Divertikel der Gallenblase, Klin. Wchnschr. **7**:216, 1928.

Bartel, J.: Ueber eine Formanomalie der Gallenblase und ihre biologischen Beziehungen, Wien. klin. Wchnschr. **31**:605, 1918.

Blakeway, H.: Congenital Absence of the Gallbladder, Lancet **2**:365, 1912.

Bower, J. O.: Congenital Absence of the Gall-Bladder, Ann. Surg. **88**:80, 1928.

Boyden, E. A.: The Accessory Gall-Bladder: An Embryological and Comparative Study of Aberrant Biliary Vesicles Occurring in Man and the Domestic Animals, Am. J. Anat. **38**:177, 1926.

Brack, E.: Ueber das angeborene Fehlen der Gallenblase, Deutsche Ztschr. f. Chir. **221**:283, 1929.

Braun, A.: Doppelbildung der Gallenblase, Zentralbl. f. Chir. **53**:1055, 1926.

Bronner, H.: Kongenitale Fundusknickung der Gallenblase, Cholecystographische Diagnose und klinische Bedeutung, Beitr. z. klin. Chir. **145**:132, 1929.

Bubenhofer, A.: Ueber einen Fall von kongenitalem Defect, Agenesia der Gallenblase, Anat. Hefte **27**:305, 1905.

zum Busch, J. P.: Die Stieltorsion der Gallenblase, München. med. Wchnschr. **74**:1099, 1927.

Carless and Wakeley, in Rose, W., and Carless, A.: Manual of Surgery, New York, William Wood & Company, 1927, p. 1175.

Cave, P.: Two Cases of Double Gallbladder, Lancet **1**:751, 1931.

Climan, M.: Duplication of the Gallbladder Demonstrated by Cholecystography, M. J. & Rec. **130**:73, 1929.

Costantini, P.: Acutissima torsione della cistifellea, Policlinico (sez. chir.) **37**: 149, 1930.

Coughlin, W. T.: Anomalous Origin of the Gallbladder, J. A. M. A. **78**:568 (Feb. 25) 1922.

Cramp, W. C.: Gangrene of the Gall Bladder from Twisted Cystic Duct, M. Rec. **87**:120, 1915.

Croudace, W. H. H.: A Case of Double Gall-Bladder, Brit. M. J. **1**:707, 1931.

Daux, F.: Le volvulus de la vésicule biliaire, J. de chir. **25**:356, 1925.

Deaver, J. B., and Ashhurst, A. P. E.: Surgery of the Upper Abdomen, Philadelphia, P. Blakiston's Son & Co., 1914, vol. 2, p. 42.

Desgouttes, L., and Ricard, A.: A propos d'une anomalie des voies biliaires, Lyon chir. **22**:823, 1925.

Dévé, F.: De quelques particularités anatomiques et anomalies de la vésicule biliaire, Bull. Soc. anat. de Paris **5**:261, 1903.

Dubs, J.: Ein Fall von sogenannter Torsion der Gallenblase, Schweiz. med. Wchnschr. **5**:93, 1924.

Eiben, T.: Ueber den angeborenen Defekt der Gallenblase, Inaug. Dissert., Giessen, O. Kindt, 1910.

Eisendrath, D. N.: Anomalies of the Bile Ducts and Blood Vessels, J. A. M. A. **71**:864 (Sept. 14) 1918.

Esau, P.: Die Wandergallenblase und ihre Stieldrehung, Arch. f. klin. Chir. **147**:192, 1927.

Ewers, T.: Ein Fall von kongenitalem Defekt der Gallenblase, Inaug. Dissert., Giessen, O. Kindt, 1914.

Feldmann: Ueber die Stieltorsion der hydropischen Gallenblase, Schweiz. med. Wchnschr. **7**:849, 1926.

Ferguson, E. D.: Surgical Malposition of the Gall Bladder, Am. Med. **2**:992, 1901.

Fifield, L. R.: Torsion of the Gall Bladder, Brit. M. J. **1**:920, 1925.

Fischer, A.: Ueber Torsion der Gallenblase, Zentralbl. f. Chir. **52**:1527, 1925. Ein weiterer Fall von Stieltorsion der Gallenblase, Berlin klin. Wchnschr. **47**:1784, 1910.

Frankau, C. H. S.: Torsion of the Gall Bladder, Brit. J. Surg. **10**:301, 1922.

Fuld, J.: Ueber angeborenes Fehlen der Gallenblase, Inaug. Dissert., Munich, 1924.

Garofalo, F.: Un caso di cistifellea doppia, Bull. d. sc. Med., Bologna **99**:380, 1927.

Gay, R. J.: Developmental Anomalies of the Gall-Bladder, with a Report of a Case, Tr. Chicago Path. Soc. **5**:108, 1902.

Giuliani, G.: Anomalia congenita dei dotti biliari e pancreatici con calcolosi, Arch. ital. di chir. **9**:537, 1924.

Goebel, cited by Kehr (1913).

Golob, M.: Interesting Aspects of a Case of Congenitally Absent Gallbladder, J. A. M. A. **89**:691 (Aug. 27) 1927.

Grunert, A.: Ueber Stieldrehung der Gallenblase, Arch. f. klin. Chir. **143**:527, 1926.

Gwynne, R. G.: Torsion of the Gall Bladder, Brit. M. J. **1**:823, 1930.

von Haberer, H.: Ueber Doppelung der Gallenblase, Arch. f. klin. Chir. **126**:25, 1923.

Haberland, H. F. O.: Studien an den Gallenwegen, Arch. f. klin. Chir. **139**:319, 1926.

Hansen, cited by Shipley.

Harris, H. A.: Intrahepatic Ectopia Testis Combined with Ectopia Vesical, Congenital Umbilical Hernia and Abnormal Gallbladder, Arch. Surg. **13**:644 (Nov.) 1926.

Hartung, A.: Linksgelagerte Gallenblase im Röntgenbild, Röntgenpraxis (Heft 11) **4**:392, 1932.

Hayes, R.: Double Gall Bladder with Double Cystic Duct, Radiology **16**:66, 1931.

Herrmann, S. F.; Hicks, G. S., and Martin, D. L.: Double Gallbladder: Report of a Case, Arch. Surg. **27**:905 (Nov.) 1933.

Hinder, H. C.: Three Cases of Abnormality of the Gall Bladder, Australian M. Gaz. **28**:435, 1909.

Hoffman, J. J., and Jackson, J. A.: Congenital Absence of the Gallbladder, New York M. J. **91**:338, 1910.

Hofmann, A. H.: Eine seltene Lage der Gallenblase, Zentralbl. f. Chir. **52**:456, 1925.

Holden, W. B.: Volvulus of the Gallbladder, J. A. M. A. **88**:1077 (April 2) 1927.

Holderman, H. H.: Double Gall-Bladder, Ann. Surg. **91**:475, 1930.

Holmes, J. B.: Congenital Obliteration of the Bile Ducts, Am. J. Dis. Child. **2**: 405 (June) 1916.

Howard, C. P., and Wolbach, S. B.: Congenital Obliteration of the Bile Ducts, Arch. Int. Med. **8**:557 (Dec.) 1911.

Huddy, G. P. B.: Torsion of the Gall Bladder, Lancet **2**:120, 1926.

Irwin, S. T.: Torsion of the Gall Bladder, Brit. J. Surg. **9**:310, 1921.

Ishiyama, F.: Ein Fall von kongenitaler Agenesie der Gallenblase mit Choledochussteinen, Arch. f. klin. Chir. **149**:183, 1927.

Jenckel: Volvulus der Gallenblase mit Gangrän, Deutsche Ztschr. f. Chir. **197**: 171, 1926.

Jonas, H. C.: A Case of Torsion of the Gall Bladder, Brit. M. J. **1**:1016, 1923.

Jones, R. W.: A Case of Imperforate Anus with Megacolon and Terminal Peritonitis, Brit. J. Surg. **13**:575, 1926.

Judd, E. S.; McIndoe, A. H., and Marshall, J. M.: Surgery of the Biliary System, in Lewis, Dean: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1929, vol. 7, chap. 2.

Kammerer, F.: Diverticulum of the Gall Bladder, Ann. Surg. **55**:320, 1912.

Kehr, H.: Eine seltene Anomalie der Gallengänge, München. med. Wchnschr. **49**:229, 1902.

Die Praxis der Gallenwege Chirurgie, Munich, J. F. Lehmann, 1913.

Keibel and Mall: Human Embryology, Philadelphia, J. B. Lippincott, 1912, vol. 2, p. 413.

Kennon, R.: A Double Gall Bladder Opening by Two Cystic Ducts into the Common Bile Duct, Brit. J. Surg. **20**:522, 1933.

Kettner, G.: Ueber einem Fall kompletter Torsion der Gallenblase, Zentralbl. f. Chir. **57**:589, 1930.

Kleiber, N.: Zur klinisch-diagnostischen Bewertung der cholecystographischen Befunde, Deutsche Ztschr. f. Chir. **222**:189, 1930.

Knox, L. C.: Congenital Absence of the Gallbladder, Proc. New York Path. Soc. **22**:166, 1922.

Krabbel, M.: Die Stieltorsion der Gallenblase, Deutsche Ztschr. f. Chir. **154**:76, 1920.

Krémer, A.: Príspevok K Cholecystografi, Bratisl. lekár. listy **12**:315, 1932.

Kubig, G.: Ueber Volvulus der Gallenblase, München. med. Wchnschr. **59**:1998, 1912.

Ladd, W. E.: Congenital Atresia and Stenosis of the Bile Ducts, J. A. M. A. **91**:1082 (Oct. 13) 1928.

Lemon, F.: Anatomical Peculiarities of a Gall-Bladder and an Appendix, Lancet **1**:1265, 1905.

Leopold, O.: Choledochussteine bei kongenitalem Defekt der Gallenblase, Deutsche Ztschr. f. Chir. **110**:625, 1911.

Lett, H.: Two Unusual Conditions of the Gall Bladder, Lancet **1**:1099, 1909.

Lintz, W.: Cholecystography and Lyon-Meltzer Test in a Patient with a Congenitally Absent Gall Bladder, Am. J. M. Sc. **173**:682, 1927.

Loughran, H. G.: Anatomical Peculiarities of a Gall Bladder, Lancet **2**:483, 1905.

Lutaud, P.: Sur un cas de torsion de la vésicule biliare, J. de chir. **25**:82, 1925.

Machmull, G.: Congenital Absence of the Gall Bladder in Man, Ann. Surg. **91**: 789, 1930.

Marinacci, S.: Torsione della cistifellea-idrope acute, Policlinico (sez. prat.) **33**: 194, 1926.

Martin, W.: Recent Controversial Questions in Gall Bladder Surgery, Ann. Surg. **79**:424, 1924.

Mayer, W.: Ein weiterer Fall von Stieltorsion der Gallenblase, Berlin klin. Wchnschr. **45**:1602, 1908.

Meade, H.: Torsion of the Gall Bladder, Brit. M. J. **1**:687, 1929.

Mentzer, S. H.: Anomalous Bile Ducts in Man, J. A. M. A. **93**:1273 (Oct. 26) 1929.

Meyer, A. W.: Doppelte Gallenblase, Deutsche Ztschr. f. Chir. **211**:404, 1928.

Aplasia der Gallenblase, Deutsche Ztschr. f. Chir. **219**:411, 1929.

Mühsam, K.: Steildrehung der Gallenblase, Berlin klin. Wchnschr. **45**:1179, 1908.

Müller, J.: Ein Fall von Aplasie der Gallenblase, Inaug. Dissert., Munich, 1920.

Murray, J. F.: Torsion of the Gall Bladder, Brit. J. Surg. **20**:687, 1933.

Nadeau, O. E.: Diverticulum of the Gall Bladder Perforating the Liver, Med. & Surg. **1**:73, 1917.

Naegeli, T.: Kongenitale Aplasie der Gallenblase, Virchows Arch. f. path. Anat. **233**:179, 1921.

Nagel, G. W., cited by Mentzer, S. H.: A Clinical and Pathological Study of Cholecystitis and Cholelithiasis, Surg., Gynec. & Obst. **42**:782, 1926.

Nehrkorn: Gangrän der Gallenblase durch Steildrehung, Deutsche Ztschr. f. Chir. **96**:319, 1908.

Nichols, B. H.: Double Gall-Bladder, Radiology **6**:255, 1926.

Niemack, J.: A Case of Choledochotomy for Cholesterin Stone with Absence of Gall Bladder, Surg., Gynec. & Obst. **7**:487, 1908.

Odermott, cited by Haberland.

Ormos, Pál: Ein Fall von Gallenblasentorsion, Gyógyászat **62**:484, 1922; Reviewed, Zentralbl. org. f. d. ges. Chir. **19**:543, 1922.

Petrina, E.: Un caso raro di duplice cistifellea con doppio dotto cistico, in uno dei quali trovasi un calcolo, Policlinico (sez. prat.) **37**:1416, 1930.

Phillips, E. W. M. H.; Isaaca, D., and Macdonald, J.: Four Biliary Anomalies: A Plea for Cholecyst-Gastrostomy, Brit. M. J. **1**:665, 1931.

Priesel, A.: Verdoppelung der Gallenblase beim Menschen, Virchows Arch. f. path. Anat. **265**:76, 1927.

Reichel: Fälle von Volvulus der Gallenblase, München. med. Wchnschr. **66**:884, 1919.

Rolleston, H., and McNee, J. W.: Diseases of the Liver, Gall-Bladder and Bile Ducts, New York, The Macmillan Company, 1929, p. 659.

Rycroft, B. W.: A Case of Bilocular Gall-Bladder, Brit. J. Surg. **10**:579, 1923.

Scammon, R. E.: On the Development of the Biliary System in Animals Lacking a Gall-Bladder in Post-Natal Life, Anat. Rec. **10**:543, 1916.

Schachner, A.: Anomalies of the Gall Bladder and Bile Passages, Ann. Surg. **64**:419, 1916.

Schmidt, L.: Zur Aplasie der Gallenblase, Deutsche Ztschr. f. Chir. **210**:409, 1928.

Schulz, J.: Ein Beitrag zur Gallensteinchirurgie, Beitr. z. klin. Chir. **92**:359, 1914.

Sherren, J.: A Double Gall-Bladder Removed by Operation, Ann. Surg. **54**:204, 1911.

Shipley, A. M.: Torsion of the Gallbladder, Arch. Surg. **14**:968 (May) 1927.

Slaughter, F. G., and Trout, H. H.: Duplication of the Gall Bladder, Am. J. Surg. **19**:124, 1933.

Stetten, D. W.: Cholecystectomy in a Case of Intrahepatic Gall Bladder, Ann. Surg. **97**:296, 1933.

Stone, I. S.: Congenital Absence of the Gall Bladder, Am. J. M. Sc. **135**:889, 1908.

Strauss, M.: Die Stieldrehung der Gallenblase, Beitr. z. klin. Chir. **122**:322, 1921.

Strukow, A. J.: Zwei Gallenblasen beim Menschen, Centralbl. f. allg. Path. u. path. Anat. **50**:420, 1931.

Sutter, A.: Die Stieltorsion der Gallenblase, Beitr. z. klin. Chir. **133**:519, 1925.

Therstappen, F. I.: Hochgradige Stieltorsion der Gallenblase, Fortschr. d. Med. **46**:125, 1928.

Toida, R.: Ueber einem Fall von grosser congenitaler sanduhrförmiger Gallenblase, Arch. f. klin. Chir. **100**:1188, 1913.

Vastine, J. H.: Diverticulum of the Gallbladder, Am. J. Roentgenol. **31**:603, 1934.

Virenque, cited by Haberland.

Wakeley, C. P. G.: A Double Gall-Bladder Removed by Operation, Brit. J. Surg. **15**:334, 1927.

Hunter, J. B., in Rose, W., and Carless, A.: Manual of Surgery, New York, William Wood & Company, 1930, p. 1205.

Walker, I. J.: Anomaly of Bile Ducts, Boston M. & S. J. **196**:733, 1926.

Walter, O. M., and Neiman, A.: Case Report of an Intrahepatic Gallbladder in an Adult with a Review of the Literature, Illinois M. J. **60**:478, 1931.

Walton, A. J.: Congenital Malposition of the Gall-Bladder, Lancet **1**:925, 1912.

Wightman: Accessory Cystic Duct, West. M. Rev. **2**:234, 1915.

Wischnewsky, A. W.: Doppelgallenblase, während der Operation aufgedeckt, Arch. f. klin. Chir. **135**:779, 1925.

Wolfson, W. L.: Supernumerary Gall-Bladder, Am. J. Surg. **6**:88, 1929.

Yoell, R., cited by Mentzer.

A REVIEW OF UROLOGIC SURGERY

ALBERT J. SCHOLL, M.D.
LOS ANGELES

E. STARR JUDD, M.D.
ROCHESTER, MINN.

JEAN VERBRUGGE, M.D.
ANTWERP, BELGIUM

ALEXANDER B. HEPLER, M.D.
SEATTLE

ROBERT GUTIERREZ, M.D.
NEW YORK
AND
VINCENT J. O'CONOR, M.D.
CHICAGO

(*Continued from p. 1042*)

URETER

Tumor.—Mathe and De La Peña [35] stated that early diagnosis of primary carcinoma of the ureter affords the only opportunity of cure by early surgical removal. The possibility of this lesion should be considered in patients who have the general symptoms of cancer, who present the triad of hematuria, lumbar pain and tumefaction of the kidney, in whom obstruction of the ureter is encountered and in whom the possibility of the presence of stone, stricture of the ureter and tumor of the kidney can be definitely eliminated. Salient points in establishing a positive diagnosis are: the presence of a tumor projecting from the orifice of the ureter or in the adjacent mucosa, an impassible obstruction in the ureter, modification of the ureteral spurt, nonperistaltic hematuria resulting from attempts to pass with a catheter, and a filling defect of the ureter, as revealed by urogram, with hydro-ureter and hydronephrosis above the point of pressure. The treatment of choice is complete nephro-ureterectomy, when feasible, including removal of the intramural portion of the ureter in one operation.

Hunter [36] reported 2 cases of primary carcinoma and 1 of benign papilloma of the ureter. The kidney and ureter were removed sepa-

35. Mathe, C. P., and De La Peña, Emilio: Primary Carcinoma of the Ureter, California & West. Med. **42**:357 (May) 1935.

36. Hunter, A. W.: Primary Tumor of the Ureter: End Results in Three Cases, J. Urol. **33**:443 (May) 1935.

rately in the first case. The kidney was enlarged, soft and definitely hydronephrotic, and the pelvis was dilated. The ureter was dilated throughout. Within 1½ inch (3.5 cm.) of the lower end of the ureter there was a sausage-shaped swelling which measured approximately 2 inches (5 cm.) in length, and ¾ inch (2 cm.) in diameter; it was moderately firm, and the bulk of this mass could be moved inside the ureter. The ureter was opened from end to end and revealed a villous tumor practically occluding the lumen. In the second case nephro-ureterectomy was performed also. The distal 5 cm. of ureter was dilated to 1.5 cm. in diameter. This portion was firm, and there was apparently a tumor either within the lumen or within the walls of the ureter. The upper extremity of this dilated portion was 8 cm. from the kidney; a grayish-red, papillomatous-appearing growth 3 cm. long and 1 cm. in diameter, of the shape of a date stone, was found; it was growing from a narrow pedicle attached to the wall of the ureter at the upper extremity of the growth, hanging downward in the lumen of the ureter. In the third case, a fusiform enlargement was found in the lower portion of the ureter, extending down to the junction of the ureter and the vas deferens. Beyond this point the ureter lessened in size until it became of normal caliber. The lower end was ligated and severed at its entrance to the bladder. The upper end was ligated at the highest point possible and cut across. The intervening portion of ureter was removed. This tumor was shown by microscopic section to be a primary papillary carcinoma of the ureter.

Primary tumors of the ureter are rare. Although secondary extensions from papillary carcinomas of the renal pelvis are common, only 61 cases of primary carcinoma have been reported.

Ravich [37] described a case of neurofibroma of the left ureter in a man aged 50, whose chief complaint was pain in the left lumbar region, with extension to the genital region. Examination gave evidence of obstruction of the left ureter in its upper third, with advanced hydronephrosis and a periureteral mass demonstrable by roentgenograms. A diagnosis was made of ureteral tumor, and at operation a mass was found surrounding and obstructing the ureter in its upper third. Section proved it to be a neurofibroma. Nephro-ureterectomy resulted in cure, for the patient was living and well at the time of the report, four years after operation.

Although neurofibromas have been reported as occurring in almost every other organ of the body, the aforementioned report is the first one of neurofibroma involving the ureter.

---

37. Ravich, A.: Neurofibroma of the Ureter: Report of a Case with Operation and Recovery, Arch. Surg. **30**:442 (March) 1935.

Stone.—Foley [38] stated that the management of stone of the ureter by expectancy and manipulation has been employed too extensively. The morbidity, mortality and hardships are underestimated, and the just purpose of such methods is often subordinated to zeal for their use. They are conservative only when they serve the patient's welfare.

Lumbar ureterotomy should always be of more minor character than pelvic ureterotomy. This fact is not properly recognized. It should always be taken into account in determining between expectancy and manipulation on the one hand and open operation on the other. Lumbar ureterotomy, as usually described and observed, is a major operation and does not properly take advantage of anatomy, toward the ends of simplicity, accuracy and minimal trauma. The improved methods for this procedure, however, take advantage of anatomy toward these ends, and this greatly minimizes the operation. A lumbar stone of the ureter, 5 mm. or more in diameter, should be removed immediately by lumbar ureterotomy. Only minute stones should be allowed to pass into the pelvic ureter for spontaneous passage or manipulative removal.

Engel [39] stated that the multiplicity of methods advocated for manipulative removal of stones of the ureter is proof of the inadequacy of any one procedure. He has employed multiple catheters, the spiral stone extractor and dilation of the ureter below the stone with bougies and has been impressed with the infrequency with which the stones have been satisfactorily removed by these means. In his experience manipulative procedures often cause sharp reactions associated with chills, fever and rather marked prostration. Manipulation may traumatize the ureter and mucosa of the ureter sufficiently to produce stricture. Additional disadvantages are the prolonged period that is usually necessary to secure passage of the stone, during which time the patient is in fear of recurrent colic, and the period of morbidity is increased by the manipulative procedures. Open operation is often necessary after the patient has been exhausted by the preliminary procedures. Open operation for removal of stones in the ureter has been favored by Engel in the majority of instances. The situation of the calculus is important and should be considered when the decision as to treatment is made.

Transplantation.—Smith [40] stated that diversion of the urinary stream is indicated in exstrophy of the bladder, in cases of intractable submucous cystitis, in certain cases of vesicovaginal fistula, in a few cases of tuberculosis of the bladder, and in selected cases of carcinoma of the bladder. Ureterostomy is particularly suitable in conjunction with total cystec-

38. Foley, F. E. B.: Management of Ureteral Stone: Operation Versus Expectancy and Manipulation, J. A. M. A. **104**:1314 (April 13) 1935.

39. Engel, W. J., in discussion on Foley.[38]

40. Smith, G. G.: Diversion of the Urinary Stream, J. Urol. **33**:179 (Feb.) 1935.

tomy. The chief complication is sloughing of the ureter. The mortality is less than with uretero-enterostomy. Uretero-enterostomy is especially suitable in case of exstrophy and vesicovaginal fistula. It is less suitable in cases of cancer of the bladder, largely because of the lowered resistance of the patient and the likelihood of dilatation of the ureter and pre-existing urinary infection. The late results of uretero-enterostomy are still sub judice. This phase of the subject has not been thoroughly studied, but a number of cases have been reported in which renal calculi and pyelonephritis have developed.

Injuries.—In the treatment of operative injuries of the ureter the problem varies with the time since the injury, the distance of the injured portion from the bladder, the involvement—whether of one or both ureters, and the type of injury; that is, whether the ureter is completely severed, completely severed with removal of a segment, partially severed, ligated or whether it has been clamped.

When confronted by this accident many surgeons ligate the proximal end of the severed ureter. This practice is to be severely condemned when circumstances are favorable for repair. Its late results are a functionless kidney or hydronephrosis; its immediate complications may be urinary leakage and extravasation, peritonitis, urinary fistula or renal infection necessitating further surgical intervention.

Sisk [41] has used a simple method of immediate repair which was independently described by Young. A ureteral catheter as large as will freely move through the ureter without interfering with the circulation is introduced into the ureter and passed into the renal pelvis. Introduction may be made through a cystoscope, or the catheter may be passed each way from the point of injury, the lower end being brought out through the urethra later on. Over this catheter, end-to-end anastomosis is made with fine catgut, the sutures being passed through the serous and muscular layers. With satisfactory drainage the catheter may be left in place for many days to prevent contraction of the lumen during healing.

Exceptions are to be made (1) when the injury is sufficiently low to permit reimplantation into the bladder, which is preferable; or (2) when a segment of sufficient length to cause tension has been removed. If there is tension satisfactory healing will not occur, and implantation into the bowel is the operation of choice.

In cases of old injuries complete studies are necessary to determine the advisability of plastic repair, of implantation into the bladder or bowel, of nephrostomy or of nephrectomy.

---

41. Sisk, I. R.: Operative Injuries of the Ureter, Surg., Gynec. & Obst. **60**: 857 (April) 1935.

Sisk reported 5 cases of operative injury of the ureter; in the first of these the operative repair described was successful, as was proved by re-examination five years after the injury.

Follow-up studies are necessary, for absence of symptoms is no criterion of a satisfactory result.

Physiology.—Ockerblad, Carlson and Simon [42] studied the effect of morphine on the intact ureters of 24 patients. They made hydrophorographic tracings by Trattner's method, and some of the patients were subjected to roentgen examination. Morphine given subcutaneously in the usual clinical doses was found to increase markedly ureteral tone and the amplitude of the contractions; the larger the dose, the greater the effect. The effect is produced by morphine in from two to five minutes and persists for at least three hours and probably longer. Atropine in doses of $\frac{1}{100}$ grain (0.0006 Gm.) invariably eliminates the contractions of the ureter that is stimulated by morphine with a consequent loss of tone but does not act strikingly or constantly when given alone. The belief that morphine quiets the ureter is incorrect; instead, it acts as a stimulant.

Compression.—Baker and Lewis [43] compared the urographic outlines as obtained for pregnant women and for patients with tumor of the pelvis. They found a high degree of dilatation of the upper part of the urinary tract in pregnant women, in patients with large ovarian cysts and in patients with fibromas of the uterus. Dilatation of the right ureter and pelvis was much greater and more common than dilatation on the left side. The investigators are of the belief that the cause of dilatation is mechanical pressure and that the reason dilatation in the left tract is not as great as in the right is that the sigmoid acts as a cushion to protect the left ureter from direct pressure. The sigmoid causes torsion of the uterus with resultant increased pressure on the right ureter.

BLADDER

Carcinoma.—Gehrmann [44] stated that the tumor of the bladder resulting from the handling of aniline is an entirely new occupational disease in this country; the first cases appeared in 1931. The patients who were examined had been engaged in the manufacture of dye intermediates. There was a total of 49 tumors—35 papillomas and 14 carcinomas. The incidence of tumor of the bladder was 4.1 per cent in the group of patients examined.

---

42. Ockerblad, N. F.; Carlson, H. E., and Simon, J. F.: The Effect of Morphine upon the Human Ureter, J. Urol. **33**:356 (April) 1935.

43. Baker, E. C., and Lewis, J. S., Jr.: Comparison of the Urinary Tract in Pregnancy and Pelvic Tumors, J. A. M. A. **104**:812 (March 9) 1935.

44. Gehrmann, G. H.: Papilloma and Carcinoma of the Bladder Among Dye Workers, Surg., Gynec. & Obst. **60a**:555 (Feb.) 1935.

Chemicals producing tumor are encountered in the intermediate stage of manufacture. Careful analysis indicated that beta naphthylamine, benzidine and alpha naphthylamine are the causative materials. In repeated examinations of a group of patients exposed to aniline only over a period of eighteen years there was no incidence of tumor; this makes it seem possible that aniline is at least a questionable factor. The period of exposure varied from five to twenty-three years, the average being eleven years. German observers have reported 1 case in which the tumor appeared after as short a period of exposure as two years; there was no evidence in this instance, however, to indicate that the patient had a normal bladder when he began to work with the chemicals. Age does not seem to bear any direct relationship to incidence. In this group 1 patient was 24 years of age; 9 were from 30 to 40; 13 were from 40 to 50, and 12 were from 50 to 60 years old.

Absorption takes place by the respiratory, gastro-intestinal and cutaneous routes. The most important route is the respiratory one, but it should be borne in mind that cutaneous absorption of intermediates can, under conditions of careless handling, become the most significant portal of entry.

Gehrmann stated that to prevent the disease the manufacture of dye intermediates must be conducted in such a manner as absolutely to exclude dust, fumes and contact of offending substances with the skin. This means that there must be an entirely closed process for the manufacture of alpha and beta naphthylamine, benzidine and aniline. Tumors classified as of grade 1 malignancy, which are small and easily accessible, are treated by bipolar fulguration through the cystoscope. Large tumors of grade 1 are treated by open operation. Tumors of grade 2 malignancy, which are reasonably small and easily accessible, are treated through the cystoscope by fulguration. Open operation should be performed in cases of large and inaccessible tumor. Open operation and implantation of radon seeds is the method to be used in treatment of all tumors of grade 3 or 4 malignancy.

Pfahler and Vastine [45] stated that tumors of the bladder varying from 1 cm. in diameter to a large size can be demonstrated clearly by means of pneumocystograms. Progressive enlargement or progressive reduction in size of this lesion can be shown and recorded photographically. The method of examination is comparatively painless and harmless. During treatment by high voltage roentgen therapy the progressive disappearance of the tumor may be recorded serially from time to time.

---

45. Pfahler, G. E., and Vastine, J. H.: The Roentgen Diagnosis and Treatment of Tumors of the Bladder: Their Serial Study with Pneumocystograms, Showing Results of Treatment by Irradiation, J. A. M. A. **104**:609 (Feb. 23) 1935.

Barringer [46] stated that up to a year ago there were two methods of treatment of cancer of the bladder. One was removal by operation; the other was application of radium suprapubically or through the cystoscope. Barringer favors treatment with radium. It can be used in cases of inoperable tumor with some degree of success. There are some tumors of grade 4 malignancy that are not satisfactorily attacked by either operation or radium therapy. Barringer has had only 1 patient with a tumor of grade 4 malignancy who remained well for eight years after treatment. Tumors of this grade should be irradiated externally before anything is done, after the specimen and the cystogram have been examined. If desired results are not obtained by external irradiation this should be supplemented by some form of internal irradiation. It is Barringer's belief that extensive tumors of grade 3 malignancy, difficult to remove or to irradiate, should be attacked by preliminary external irradiation. That method should be employed in the future, and, with careful application of external irradiation, it should add 5 per cent to the number of five-year cures.

Ferguson [47] reported that in 159 of 902 cases entered in the Bladder Tumor Registry, pneumocystograms were made. The procedure was said to be of value in the diagnosis of the lesion in 108 of these cases. A roentgen technic, favored in the treatment of carcinoma of the bladder, is the method of divided doses. Carcinoma of the bladder does not usually yield to a tissue dose of less than eight or ten erythema doses. If any such dose is to be delivered by external means of irradiation it must be by the technic of divided doses. This has been done successfully within the last eighteen months by Dean. The most satisfactory results seem to be obtained when it is known in advance that the malignancy of the tumor is either of grade 3 or grade 4. Carcinomas of grade 1 and grade 2, especially those that contain squamous pearls, are radioresistant and do not yield to the average dose that can be delivered into the bladder in this manner.

Ferguson advocated ascertaining the possibilities of roentgen treatment of carcinoma of the bladder, making a biopsy in every instance, utilizing the Coutard technic or some modification of it in delivering tissue doses adequate to carcinomas of grades 3 and 4 and avoiding treatment by this method of tumors of grade 1 or 2.

Urinary Incontinence.—Abeshouse [48] stated that incontinence of urine is an uncommon, although not an infrequent, complication of oper-

---

46. Barringer, B. S., in discussion on Pfahler and Vastine,[45] J. A. M. A. **104**: 613 (Feb. 23) 1935.

47. Ferguson, R. S., in discussion on Pfahler and Vastine.[45]

48. Abeshouse, B. S.: Repair of Urinary Incontinence Following Prostatectomy by Transplantation of the Gracilis Muscle: Report of Two Cases, J. Urol. **33**:28 (Jan.) 1935.

ation on the prostate gland. It occurs more frequently after perineal than after suprapubic prostatectomy, or resection of the prostate gland. Development of this condition following prostatectomy is attributable to total destruction of derangement of both the internal and the external sphincter of the bladder. In cases of complete or partial incontinence following this operation, various anatomic defects or pathologic lesions about the posterior urethra and neck of the bladder may be associated with loss of function of both sphincters.

Satisfactory results have been obtained in treatment of this condition, in female and in male patients, by transplantation of the gracilis muscle. In the 2 cases of complete incontinence of urine following perineal prostatectomy reported by Abeshouse, the operative technic employed was originally described by Player and Callander. In one instance complete control of urine during the day and the night was restored. In the other case complete control of urine was restored during the night and in the reclining posture, but there was partial diurnal incontinence when the patient assumed the upright position and after excessive muscular effort. It is important and necessary that the urine be diverted from the field of operation. Drainage of urine through an indwelling urethral catheter may suffice in certain types of incontinence of female patients, who are treated by extravesical plastic operations on the sphincters of the bladder and urethra or by transplantation of a voluntary muscle or fascia. However, this type of drainage is usually inadequate and inefficient in similar cases of incontinence of male patients. Drainage of urine by means of suprapubic cystotomy, performed preferably several days previous to transplantation of the muscle, has the advantages of permitting excoriation of the skin about the external genitalia to clear up, minimizing the subsequent influence of such cutaneous lesions on development of an infection in the region of the operation, keeping the operative field dry during and after transplantation of the muscle and facilitating primary healing by eliminating the irritative and infectious tendency of an indwelling urethral catheter.

Deming [49] stated that the fact that 9 cases of simple transplantation of the gracilis muscle have been reported by six different surgeons, with good results in 8 and no deaths, indicates that this plastic procedure has definite merit. The 1 failure was attributable to incorrect application of the procedure and not to the operation itself. The indications for use of the method may be considered to be present when the sphincters of the bladder are absent or vary from normal and there is a good detrusor urinae muscle of the bladder. It should not be used when the detrusor

49. Deming, C. L., in discussion on Watson, E. M.: Complete Rupture of the Urethra, J. Urol. **33**:73 (Jan.) 1935.

urinae muscle is atonic or when lesions of the spinal cord, either congenital or acquired, render the musculature of the bladder inactive. Other muscles, such as the pyramidalis muscle, levator ani muscle and abductor longus muscle have been used for sphincteric action, but they are not as satisfactory for transplantation as the gracilis muscle. Dispensing with the normal use of this muscle is not a detriment to the patient. The muscle has a long, slim belly and is readily approached. It has a double supply of nerves and blood. Its upper two-thirds is supplied by the obturator nerve, which comes from the direction of the origin of the muscle and lies in the direction of the desired transplantation, so that it can be turned upward without injury to the nerve and blood vessels. The sex of the patient is not important except that there is more difficulty in exposing the urethra of the male. Tunnelling of the tissues for a few centimeters near the urethra is preferable to complete incision. Either the right or left muscle may be used; however, it seems that a right-handed person probably would be more conscious of the use of the right gracilis muscle, and adaptation of the new use of the muscle would be accelerated.

Perineal Drainage.—Illyès [50] uses perineal drainage after resection of the bladder, diverticulectomy, ureteral implantation or any operation on the bladder in which perivesical leakage or infection is likely to occur. This does not include transvesical prostatectomy, in which procedure, after the enucleation, Illyès swabs the prostatic cavity with 30 per cent solution of silver nitrate and then packs it.

A blunt forceps is pushed under the symphysis pubis, to either side of the midline, penetrates the triangular ligament and emerges in the anterior triangle of the perineum. The drainage tube is drawn upward into the pelvis. This is done in both male and female patients, and with care injury to important structures can be avoided.

Holding upward the concave side of the slightly bent blunt forceps, the surgeon feels the lower part of the symphysis pubis and perforates the pelvic fascia 1¼ inch (3 cm.) from the midline, near the lower edge of the pubic bone. He presses down the inner end of the instrument toward the abdominal cavity in order that this end may remain in a straight line as much as possible, directly under the pubic bone. Thus he avoids injuring the urethra as well as the pudendal artery and nerve. At the sharp angled union of the two sides of the pubic bone under the symphysis pubis and the ligamentum arcuatum, respectively, he finds the lamina intercruralis connecting the two sides of the acute angle, in which are arteries and veins running to the corpus cavernosum. This

50. Illyès, Góza: Perineal Drainage for Certain Bladder Operations, Brit. J. Urol. **7:1** (March) 1935.

part could be injured only in the event of forcing the outer end of the instrument up toward it, which is well-nigh impossible because the surgeon could not depress the hand holding the instrument far enough toward the abdominal cavity.

This method of drainage has been of great help in numerous cases, and if there is no longer need for draining, the tube can be taken out. The channel of from 3 to 4 mm. diameter left by it contracts entirely within a few days and closes without leaving the slightest tract.

Use of Catheter.—Wesson [51] stated that an overdistended bladder, in which there is no obstruction, will never tear without external violence; hence, there is no danger of a cord bladder rupturing spontaneouly. With a small quantity of morphine, and patience, the bladder will overflow and become automatic within ninety-six hours. Wesson cited 22 cases which illustrate the increased morbidity and financial cost resulting when the problem is complicated by infection from the use of a catheter. He does not consider it advisable to convert a typical neurosurgical case into a urologic one by the routine use of a catheter.

VESICOVAGINAL FISTULA

Kirwin and Lowsley [52] reported a study of treatment in 60 cases of vesicovaginal fistula as seen at the New York Hospital between 1844 and 1933 and concluded that this lesion is one of the most difficult to cure known to surgery. The first satisfactory technic was devised by Marion Sims in 1852, and that procedure, with slight modification to take advantage of certain new methods, is applicable at present.

Kirwin and Lowsley reported the case of a patient treated at the Brady Foundation for Urology of the New York Hospital, the sixty-first in a series from that hospital. The fistula was complicated by complete eversion of the bladder into the vagina, so that the everted bladder protruded at the vulva. The condition was treated in two stages. The features of the technic to be emphasized are the excision of all devitalized tissue, the bladder being incised almost to the edge of the fistula; the silver wire sutures used were similar to those used by Sims eighty years ago, and the suprapubic counterdrainage, with suction, permitted healing of the freshened edges of the fistula, which were uncontaminated by contact with urine.

---

51. Wesson, M. B.: Is the Use of a Catheter Ever Justified in Fractures of the Spine? Urol. & Cutan. Rev. **38**:572 (Aug.) 1934.

52. Kirwin, T. J., and Lowsley, O. S.: Radical Relief of Vesico-Vaginal Fistula: Report of an Unusual Case of Eversion of the Bladder Through the Fistulous Opening, and a Review of Sixty Cases Seen at New York Hospital During the Past Ninety Years, J. Urol. **33**:51 (Jan.) 1935.

PROSTATE GLAND

Obstruction.—Thompson [53] reported that 630 patients with obstruction by the prostate gland were treated by the transurethral method at the Mayo Clinic in 1934. This is an increase of 39.6 per cent over the number of patients having the same operation in the preceding year. Included in these 630 cases, in which a total of 696 transurethral resections of the prostate gland were performed, are 23 in which the patients had submitted to the same operation at the Mayo Clinic in former years. In 5 of these cases the original growth was malignant, and recurrence could be expected. In the other 18 cases, in which recurrence was not expected, it was necessary to remove 8.2 Gm. of tissue, on the average, in order to relieve the symptoms; in 7 of these cases the total weight of the tissue removed at both operations exceeded 25 Gm. These patients, on their return, refused to undergo any procedure other than transurethral resection. Because of the increased risk and longer hospitalization, prostatectomy was not urged. In each case of recurrence the stay in the hospital required for resection of the gland was less than a week, and there were no fatal cases.

The average weight of tissue removed per case during the year was 11.6 Gm.; in 73 cases the amount exceeded 25 Gm. The average period of hospitalization was shorter than in former years; 58.1 per cent of the patients stayed one week or less, an additional 28.9 per cent left in the second week, and only 13 per cent remained longer. The average period of hospitalization was eight and one-tenth days. The period of hospitalization for the group as a whole was shortened by reduction in the number of cases in which preliminary suprapubic cystostomy was performed; in 21 cases such an operation was performed at the clinic, and in 12 cases cystostomy had been performed elsewhere. Thus, in a total of 5.2 per cent of the group, as compared with 12.2 per cent in the two preceding years, preliminary suprapubic cystostomy was performed.

The painful complication of epididymitis has been almost entirely eliminated by strict attention to methods of drainage by urethral catheter rather than by routine vasectomy. The latter operation was performed in only 24.6 per cent of the cases. Epididymitis occurred in only 8 of the 630 cases studied in 1934—an incidence of 1.3 per cent. Litholapaxy was performed at the same time as resection in 28 cases, and in 7 others a tumor of the bladder was removed transurethrally at the time resection of the prostate gland was performed. There were only 2 deaths—a mortality rate of 0.3 per cent for the series of 630 cases.

---

53. Thompson, G. J.: Transurethral Surgery in 1934, Proc. Staff Meet., Mayo Clin. **10**:220 (April 3) 1935.

Carcinoma.—Barringer [54] stated that a comprehensive review of carcinoma of the prostate gland is most confusing and that it is practically a fatal disease. In 351 consecutive cases there were but 16 (4.5 per cent) carcinomas which might be called reasonably small that were confined to the region of the prostate gland and to the periprostatic region. Twenty-six (7.4 per cent) of the carcinomas were classified as of medium size, that is, reaching well beyond the prostate gland proper, but not too large and entirely within reach of the examining finger. In the remaining 309 cases (88 per cent) the carcinomas were extensive; that is, the tumor itself was large or, if the tumor was not large, the extensions around the seminal vesicles or to the lateral pelvic walls were definite, the bladder was secondarily involved, the tumor arose postoperatively or metastasis to bone or other structures was present. However, according to Barringer, there are some rays of hope; 29 (little more than 8 per cent) of his patients were well for periods varying between three years and seventeen years and ten months. The prostatic condition was such as to make one believe that the carcinoma was controlled. Nine patients went from three to five years without recurrence; 16 had no recurrence in from five to ten years, and 4 were well for more than ten years. Time alone will prove whether all these patients are cured. Transurethral resection of the prostate gland has made a great advance in coping with urinary retention, while irradiation, by radium, of the prostate gland has developed and become sufficiently accurate to enable one to irradiate a prostate gland without severe pain to the patient. External irradiation in sufficient quantity to be of any use has been employed only in the last few years. Cases of prostatic carcinoma are divided, for purposes of treatment, into classes. In the first class are those in which there is a reasonably small carcinoma of the posterior lobe with, as far as can be determined, no involvement of the other lobes and no urinary retention. The patients should receive irradiation by gold-screened needles inserted through the perineum into the prostate gland. They should, in addition, receive external irradiation. The two courses of irradiation are given as nearly simultaneously as possible. In the second class are cases in which urinary retention is the dominant symptom and in which there is involvement of the so-called median or of the lateral lobes. In these cases a suprapubic opening should be made, all obstructing portions of the prostate gland being cut out with electric cautery and gold seeds being implanted into the entire cancerous region, in exactly the same manner as gold seeds are implanted in vesical carcinomas. When the suprapubic wound heals the posterior lobe has to be carefully watched by means of palpation and possibly biopsy of aspirated material. If any

54. Barringer, B. S.: Prostatic Carcinoma, J. Urol. **33**:616 (June) 1935.

suggestion of carcinoma remains gold-screened radon perineal needles are used. Implantation of gold seeds through the perineum is ineffective because the gold seeds cannot be implanted with any degree of accuracy. After the patient has recovered from the foregoing procedure external irradiation should be given. The third class consists of cases in which the prostatic carcinoma is so enormous that it seems impossible to control. External irradiation alone is given, and if urinary retention is a dominant symptom transurethral resection should be performed. The fourth class contains cases in which there is metastasis to bone or metastasis around nerves in which amelioration is to be expected. In these cases external irradiation alone should be used. It is surprising how many years the lives of patients with metastasis to bone can be prolonged by this method.

Graves and Militzer [55] presented a clinical review of 81 cases of carcinoma of the prostate gland with metastasis and made a preliminary report of a pathologic study of the paths of dissemination in cases of this disease. In all but 6 cases of the entire series, metastasis to bone was found either by roentgen examination or at necropsy. The pelvis and sacrum were involved in 85 per cent of the cases, and the lumbar part of the spinal column was affected in 59 per cent. Pathologic fractures were observed in 5 instances.

Treatment of the local condition was necessarily limited to measures of palliation. The plan of treatment in cases of carcinoma of the prostate gland with metastasis cannot be standardized, and the procedure adopted in each case must vary with the needs of the individual patient. Transurethral resection sometimes forestalls the need for more extensive operation. High voltage roentgen irradiation and radium therapy proved to be the most effective agents for the relief of pain caused by metastasis.

Postmortem examinations were made on 74 per cent of the patients who died in the hospital. Metastasis other than in the bone was found most frequently in lymph nodes, in the lungs and in the liver. Pyelonephritis was the direct cause, or the most important contributing cause, of death in 41 per cent of the cases. It was observed clinically that advanced renal infection may exist without significant pain and tenderness in the region of the kidney. Extensive pyelonephritis may be present without marked elevation of the nitrogen content of the blood. The test of renal function with phenolsulphonphthalein frequently affords a more accurate picture of the degree of damage to the kidney than do chemical examinations of the blood.

---

55. Graves, R. C., and Militzer, R. E.: Carcinoma of the Prostate with Metastases, J. Urol. **33**:235 (March) 1935.

Rich [56] reported that in 292 consecutive postmortem examinations of men aged 50 or over who died of a wide variety of causes at the Johns Hopkins Hospital during the past three years, frank carcinoma of the prostate gland was observed in 41 cases (14 per cent) in the microscopic section taken as a routine at necropsy. Thorough examination of each gland undoubtedly would have disclosed a larger number of these tumors, many of which were so small that they were not seen macroscopically at necropsy. The report indicates, however, that carcinoma of the prostate gland occurs more frequently than is ordinarily supposed. In 65.8 per cent of these 41 cases the tumor was not recognized clinically, having been in most instances too small to have produced symptoms or to have attracted attention on physical examination. The most common site of the tumors was near the outer margins of the gland, and, even when they were only a few millimeters in size the tumors showed a tendency to invade the capsule.

Moore [57] reported preliminary conclusions concerning unrecognized carcinoma in 375 prostate glands and discussed the morphology in 52 cases of this disease. The clinical diagnosis was not made in all instances; There was no distant metastasis, and the disease was limited to one anatomic lobe of the gland. In the material on incidence an additional 11 cases of manifest carcinoma of the prostate gland are included, making a total of 63 cases of carcinoma occurring in the 375 prostate glands.

Moore's conclusions were that carcinoma of the prostate gland occurs more frequently with increasing age until 29 per cent of men who are examined at necropsy after having lived into the ninth decade have the lesion. The disease is intimately associated with senile atrophy, and the growth probably results from epithelial cells which have previously undergone atrophy. It is predominantly a lesion of the posterior lobe of the gland, but it may arise in any portion of it. Carcinoma and benign enlargement of the prostate gland are two distinct diseases which occur independently. Malignant degeneration rarely occurs in acini already hyperplastic and involved in benign enlargement. Perineural lymphatic invasion in the capsule of the prostate gland is one of the earliest changes in carcinoma, but invasion of the seminal vesicles and distant lymphatic invasion are late manifestations.

Physiology.—Deming, Jenkins and van Wagenen [58] stated that atrophy of the true prostate gland has been demonstrated after castra-

56. Rich, A. R.: On the Frequency of Occurrence of Occult Carcinoma of the Prostate, J. Urol. **33**:215 (March) 1935.

57. Moore, R. A.: The Morphology of Small Prostatic Carcinoma, J. Urol. **33**:224 (March) 1935.

58. Deming, C. L.; Jenkins, R. H., and van Wagenen, Gertrude: Some Endocrinological Relationships of Prostatic Hypertrophy: Clinical and Experimental Studies; Preliminary Report, J. Urol. **33**:388 (April) 1935.

tion. There is no evidence that castration causes atrophy of the so-called benign overgrowth of the prostate gland. The anatomic origin of prostatic hypertrophy has been definitely established. Studies have disclosed absence of submucosal glands of the prostatic urethra and trigon in the monkey; hence there is less probability of the development of prostatic hypertrophy in this animal. The rat has all the anatomic structures that might serve as origins for hypertrophy of the prostate gland. A case in which hypertrophy of this gland developed from the submucosal glands of the posterior urethra of an old rat is reported.

Stone.—Thompson and Cook [59] stated that chronic prostatitis often persists because of infected pockets or diverticula that drain only through a small prostatic duct. Treatment by ordinary methods, such as massage, irrigation, injection of antiseptic substances or diathermy, results only in temporary relief of symptoms. Surgical treatment of these structures by the transurethral route insures adequate drainage and subsequent improvement in a large percentage of cases. The cavities of the prostate gland must be widely excavated in the form of a saucer, and, if necessary, tissue must be excised to provide free flushing at the time of urination; otherwise infection will persist. Calculi embedded in the tissue of the prostate gland, which occur either primarily or secondarily to infection, can be removed by transurethral operation.

PENIS

Epithelioma.—Dean [60] presented a clinical study of 120 cases of epithelioma of the penis in which radium and roentgen therapy were used and considered the review significant in view of the fact that sufficient time had elapsed to determine to a fair degree the value of these agents. Married men predominated in this series in the ratio of 5:1. Occupation and previous constitutional diseases (excluding syphilis) seemed to be of no etiologic importance. Thirty-four per cent of 70 patients had had syphilis, and the syphilitic patients, as a group, were 8 years younger than the nonsyphilitic patients when the symptoms of cancer appeared. No Jews were found in the series. Only 4 per cent of 93 patients had a freely retractile prepuce. At least 65 per cent had some mechanical difficulty in retracting the prepuce. Thirty-seven per cent of the patients had been operated on to expose the glans penis. The flat type of cancer of the penis occurred more frequently than the papillary type. The two types seem to grow at the same rate, but the flat tumors metastasize earlier. The inguinal nodes were the site of the earliest metastasis,

59. Thompson, G. J., and Cook, E. N.: Chronic Prostatitis and Prostatic Calculus: Treatment by Incision with the Electrocautery, J. A. M. A. **104**:805 (March) 1935.

60. Dean, A. L., Jr.: Epithelioma of the Penis, J. Urol. **33**:252 (March) 1935.

but in further advanced conditions metastasis was widespread. There was an average delay of fourteen months between the onset of symptoms and the first visit to a physician. In addition, there was an average delay of twelve months between the first visit to a physician and the first examination at the Memorial Hospital for the Treatment of Cancer and Allied Diseases, making the average total delay more than two years.

Because there are several lesions which may resemble cancer of the penis the diagnosis should always be based on histologic examination. Seventy-six per cent of the patients had regional adenopathy at the first examination; of the involved glands 50 per cent were cancerous. Biopsy of aspirated material was performed as a check on physical examinations. Twenty-four per cent of the patients did not have inguinal adenopathy at their first examination. Since metastasis did not develop subsequently in any of these cases, management of the primary tumors must have been efficient.

The prophylactic treatment of cancer of the penis consists in circumcising all male infants a few days after birth. When the primary tumor was superficial and no larger than 2 cm. in diameter treatment with the radon plaque was successful in all cases. When the primary lesion was larger and had penetrated deeper, amputation was performed 1.5 cm. proximal to any visible or palpable evidence of degeneration. Of 51 patients, 64.6 per cent treated by the aforementioned methods are free from disease. Forty patients with inguinal metastasis were treated by surgical removal with or without irradiation. Five per cent are now free from evidence of cancer. It is recommended that operation for inguinal metastasis be delayed until the primary tumor has been removed and infection in the nodes has subsided. No type of external irradiation used by Dean was of value in treating inguinal metastasis from cancer of the penis.

Wolbarst [61] stated that there is no authentic recorded case of cancer of the penis in a Jew ritually circumcised in early infancy. Almost every writer on the subject has made the same comment, but the importance of this fact is in making it a convincing argument as to the prophylactic value of circumcision in the campaign against cancer of the penis and cancer generally. There is still considerable opposition to infantile circumcision by otherwise progressive physicians; they seem to prefer to wait until an existing phimosis becomes troublesome before considering circumcision, and by that time the precancerous lesion may be, and often is, already present. To be effective completely as a prophylactic measure against cancer, circumcision must be performed in early infancy. The point is often made that the freedom of Jews and Mohammedans from cancer of the penis is attributable to racial immunity, but the

61. Wolbarst, A. L., in discussion on Dean.[60]

inherent weakness of this contention has been shown by the work of Sorsby and others and also by the case of cancer in an uncircumcised Jew which Wolbarst reported in 1932. So far as can be ascertained, this is the only reported instance of cancer of the penis in a Jew. If he had been born with racial immunity it must have become seriously attenuated by virture of his retained foreskin.

Hypospadia.—Cabot, Walters and Counseller [62] reported on 38 cases of hypospadia in which operation was performed between May 1, 1930, and May 1, 1934. Besides the various procedures directed toward correction of the deformity, three general methods of constructing a urethra were principally used: the lateral flap method, a development of the old principles of Duplay and Thiersch (26 cases); the Bucknall method, and the Ombrédanne method. The operations should be divided into two groups, those concerned with correction of the deformity of curvature and those concerned with construction of the urethra. Procedures intended to overcome the deformity of curvature should be undertaken at an early age, preferably between 1 and 2 years. The smallness of the parts is not considered a handicap to this phase of the operation. The optimal time for building of the urethra is after the child has attained reasonable growth and yet some years before puberty. This procedure is best undertaken some time in the sixth year, three or four years after the deformity has been corrected. In most of the methods employed diversion of the urinary stream will be desirable. This is not necessary in the Ombrédanne operation but is essential in operations of the modified Duplay and Thiersch type. In the experience of Cabot, Walters and Counseller drainage through a perineal boutonnière made at the completion of the operation of repair has been satisfactory.

At present operations for hypospadia involve four types of procedure. There is the parallel flat type in which the urethra is made by turning in flaps with broad bases cut parallel to the normal course of the urethra and so planned as to avoid superimposition of the suture lines in the two layers. All modifications of this type are from the principles originally enunciated by Duplay and by Thiersch. It is significant that the flaps utilized in this procedure contain no hair-bearing skin in any portion. The second type involves the principle of the pedicle flap, these flaps being taken from more or less distant regions and manipulated at various stages of the procedure so as to construct a urethra. This method has been used with skill by Blair and others. In the third type, scrotal flaps are used. The typical procedure of this group is the Bucknall operation. The inner layer of the urethra is formed from the tissue extending backward from the abnormally placed

62. Cabot, Hugh; Walters, Waltman, and Counseller, V. S.: Principles of Treatment of Hypospadias, J. Urol. **33**:400 (April) 1935.

meatus, and to the extent that this lies behind the penoscrotal angle it will involve a hair-bearing area. The scrotal and preputial type is the fourth method, of which the Ombrédanne operation is the most modern example. The posterior portion of the canal is constructed from tissue coming from the scrotum, as in the Bucknall operation. The anterior portion, to a varying extent, is made from the tissue of the redundant prepuce. In this operation the posterior portion of the canal in the penoscrotal type, and particularly in the perineal type, of case draws its inner layer from scrotal, and consequently hair-bearing, tissue.

The method of parallel flaps probably produces the best restoration of the canal. It employs tissue free from hair and of fine texture, particularly well suited to its changed environment. The formation of fistula has been a relatively common complication in this type, requiring further operation for its repair, but it can generally be avoided by adherence to the principles of plastic surgery. These involve the union of tissue on relatively broad surfaces and never union of the edge-to-edge type. They also require careful hemostasis and care in regard to a thoroughly adequate blood supply. However, the blood supply of flaps thus fashioned is better than that available in most of the other operations. It is the belief of the authors that at the hands of experienced surgeons the operation gives satisfactory results. The Bucknall operation was preferred by one of them for more than ten years. It has the advantage of being almost fool-proof, and with modest experience great certainty exists that a full length canal, free from fistula, will be obtained as the result of a two-stage operation. Owing to the growth of hair in these urethral flaps the procedure has been abandoned, as this complication is considered unavoidable and introduces an element of late unsatisfactory results.

Ombrédanne reported a series of 392 cases in which his method was employed. Lyle reported 9 cases; all the patients were children, and they were not followed into adult life. The procedure does not require diversion of the urinary stream. It is apparently free from any marked risk of fistula and creates a urethra of full length and adequate caliber. It is subject to the objection inherent in the Bucknall type of operation, namely, the subsequent development of hair with formation of stone and urethral irritation attributable to the fact that some of the skin utilized for the urethra comes from a hair-bearing area. According to the experience of Cabot, Walters and Counseller the operation is free from unusual technical difficulties, and it does create a satisfactory urethra without important danger of the formation of fistula.

Absence of the Penis.—Drury and Schwarzell [63] reported a case of congenital absence of the penis of a boy aged 13.

63. Drury, R. B., and Schwarzell, H. H.: Congenital Absence of Penis, Arch. Surg. **30**:236 (Feb.) 1935.

There were no subjective symptoms, and the patient's physical and mental development were those of the average boy of his age. The testes, spermatic funiculi and vasa deferentia were normal. Urine was discharged from the rectum three or four times a day. Although excretory urography gave evidence of a bilateral hydro-ureter and some pelvic dilatation there were no symtpoms of urinary infection.

This condition is extremely rare. In the 7 cases previously recorded accompanying congenital defects were common, and ectopic erectile tissue was frequently noted. This patient had no other demonstrable abnormalities. This is the first case reported in which the urinary tract has been visualized by roentgenograms.

TESTIS

Cryptorchidism.—Thiessen and Walters [64] reported on 96 cases of undescended testis in 72 patients operated on at the Mayo Clinic, in which treatment was by temporary fixation of the testes to the thigh. The Torek method of operation was used in all these cases, and satisfactory results were obtained in 92 per cent of them. In 73.9 per cent of the cases an indirect inguinal hernia, which was associated with the undescended testis, was repaired surgically at the same time. In 47.2 per cent of the cases cryptorchidism was bilateral. Of cases in which there was unilateral nondescent of the testis the right side was affected in almost three times as many instances as the left side. The right testis was undescended in 38.8 per cent, and the left testis, in 13.8 per cent of the cases. The positions of these testes were classified into four groups: The 18 (18.7 per cent) testes in the first group were found at operation to be intra-abdominal. In the second group were 3 (3.1 per cent) testes situated at the internal inguinal ring; in the third group, 40 (41.7 per cent) testes lying in the inguinal canal, and in the fourth group, 35 (36.5 per cent) testes which were situated at the external inguinal ring.

The results of the operations have been satisfactory. Seventy-six (79.1 per cent) of the testes were maintained in normal position in a scrotum of normal size. The size of 32 (33.3 per cent) of the testes increased after operation. Eight (8.3 per cent) of the testes which before operation had been small or atrophic remained in that condition. After operation the testis was found to be in the upper portion of the scrotum in 16 cases, but before operation in 10 of these 16 cases the testis had been within the abdomen. In 69 (77 per cent) of the cases results were excellent; in 13 (14.6 per cent) results apparently

64. Thiessen, N. W., and Walters, Waltman: Cryptorchidism, Proc. Staff Meet., Mayo Clin. **10**:132 (Feb. 27) 1935.

were good; in 5 cases, in all of which the testes had been intra-abdominal at operation and in which in order to accomplish the Torek procedure extreme traction on the vessels had been necessary, the results were only fair. In 2 cases, orchidectomy had become necessary, and in 7 cases no adequate check on results was obtained.

Cabot [65] discussed three methods available at present for treating cryptorchidism. The first method is to let nature take its course, in which event it is certain that in a considerable proportion of the cases no alteration in the abnormal position of the testis will take place and permanent damage to the testis will occur. Regarding the second method, recent experimental evidence suggests that the use of a hormone derived from the anterior lobe of the pituitary gland markedly affects the development of the genital apparatus and stimulates descent of the abnormally placed testis into the scrotum. The number of instances in which this treatment has been applied to children is not sufficient to warrant a conclusion, and the method should be regarded as being still in the experimental stage. It should be emphasized, however, that this method should not be used to the detriment of the child and that its use should preferably be confined to the period between 3 and 8 or 9 years of age. If the testis has not descended into its normal position by the end of that period the method should be abandoned and operation resorted to. In the third procedure for undescended testis the work of Moore modified the views in regard to what constituted a satisfactory operative result in that it required the placing of the testis in an entirely normal position and maintaining it there. This increased the importance of the Torek type of operation as compared with other methods. Operations of the Bevan type do not so consistently fulfil this indication completely and should, according to Cabot, be largely abandoned. In regard to patients with intra-abdominal cryptorchidism, in the treatment of whom the technical difficulties of obtaining normal replacement are great, Cabot's experience indicates that a two-stage operation, in which the testis is brought down to below the level of the external inguinal ring in the first stage and to a normal position in the second stage, by the application of the Torek technic, is likely to be satisfactory in many cases.

URINARY CALCULI

Experimental Studies.—Higgins [66] reported that in a high proportion of albino rats maintained on a diet deficient in vitamin A, calculi of the urinary tract developed. If deficiency in the diet was

65. Cabot, Hugh, in discussion on Thiessen and Walters.[64]

66. Higgins, C. C.: Production and Solution of Urinary Calculi: Experimental and Clinical Studies, J. A. M. A. **104**:1296 (April 13) 1935.

continued for two hundred and fifty days, 85 per cent of the experimental animals exhibited calculi of the bladder, and 42 per cent, calculi of the kidney. The spherical, light brown stones varied from 0.5 to 8 mm. in diameter. They contained calcium phosphate chiefly, with traces of carbonates and a small amount of mucoid substance. No uric acid or oxalates were detected.

Three constant changes observed in these animals, that might be associated with formation of stones, were keratinization of the epithelium of the genito-urinary tract, urinary infection and alkalinuria. The keratinization of the epithelium was noted after the diet had been deficient in vitamin A for a period of from eight to ten weeks. Urinary infection was produced in a large percentage of rats maintained for long periods on a diet deficient in vitamin A. Infection of the bladder occurred usually after thirty days, and renal infection, after from sixty to ninety days. When the experiments had progressed for from two hundred to two hundred and fifty days 72 per cent of the rats had infection of the bladder, and in 42 per cent of these animals there was a coexistent renal infection. Alkalinuria was a constant finding in rats maintained on a diet inadequate in vitamin A. By the addition of ammonium chloride to the diet the incidence of calculi was decreased. When vitamin A was added to the deficient diet the urine became acid and calculi were not produced.

It was demonstrated in earlier experiments that if vitamin A alone was restored to the diet of rats the formation of urinary calculi could be prevented. In order to determine whether stones already formed could be dissolved by the administration of vitamin A, 30 rats with calculi of the bladder or kidney were studied. Except for the addition of cod liver oil, the diet was the same as that given when the stones were produced. Twelve rats with calculi of the bladder received the deficient diet for from thirty to sixty days. In 11 of these the calculi had entirely disappeared, as evidenced by roentgenograms, after 2 drops of cod liver oil had been given daily for a period of from fifty to sixty days. In a second group of 11 rats which had received the deficient diet for from ninety-six to one hundred and thirty days calculi of the bladder could be demonstrated roentgenographically, and coexistent renal calculi were observed in 2 rats. After the rats had been on the diet high in vitamin A for ninety days, roentgenograms gave no evidence of calculi in the bladders of 6 rats. In the other 5 the calculi were definitely smaller. The third group of 12 rats had been maintained on the deficient diet for one hundred and eighty days. Vesical calculi had been demonstrated in all these rats, and renal calculi were present in 2 of them. In four animals the calculi of the bladder had entirely disappeared one hundred and one days after cod liver oil had been added to the diet.

In the remaining rats in this series the calculi were definitely smaller. In one the renal calculi also had disappeared. Cystitis, hemorrhage under the mucosa of the bladder, and renal infection were commonly observed in this group. Keratinization of the epithelium was present to some degree in the genito-urinary tracts of all the animals in this series. A reparative process was evident in the epithelium of the ureters, kidneys and bladder, especially when there was not a marked degree of infection.

These experiments show that with the addition of vitamin A to the diet there is a tendency toward restoration of the normal epithelium along with solution of the calculi. In the first experiments at the Cleveland Clinic the calculi produced in rats on a diet deficient in vitamin A were composed chiefly of calcium phosphate; hence it seems logical to assume that the calculi in this series of experiments were of the same composition. An acid ash diet high in vitamins, especially in vitamin A, is suggested as a prophylactic measure to prevent the recurrence of calculi in patients who have had urinary lithiasis, and the rôle of vitamin A in the diet to prevent primary calculous disease is important. A high vitamin, acid ash diet has produced decrease in the size of, or total disappearance of, renal calculi in patients refusing surgical intervention and in those in whom operation has not seemed indicated. In 18 collected cases in which the high vitamin, acid ash diet has been used the renal calculi have undergone solution, according to roentgenographic evidence.

Spontaneous Fragmentation.—Chauvin [67] stated that the spontaneous fragmentation of calculi within the kidney, urinary ducts and bladder, although well known from ancient times in its anatomic modalities, remains as much a mystery as ever in its pathogenic mechanism. Various theories have been proposed; the oldest on record is that of Civiale, who thought that the stones were broken by contractions of the bladder. Other writers have ascribed the fragmentation to progressive desiccation of the stone from the center to the periphery, to the progressive increase in size of the nucleus from imbibition, which was supposed to have shattered the cortex, and to the action of mineral waters. None of these explanations is entirely satisfactory, and some are actually fantastic.

To understand how certain concretions break diametrically through their entire mass it is necessary to assume that a change occurs in the size of the whole stone, both nucleus and cortex, and such a change can be explained only by a modification of the chemical composition of the stone. The calculus is constantly bathed with urine; now, the reaction and composition of this urine may change, and there is nothing to

67. Chauvin, E.: La fragmentation spontanée des calculs urinaires, Arch. d. mal. d. reins **8**:53 (Jan.) 1934.

prevent a calculus that was, for instance, originally of uric acid, from becoming a uratic stone if the urine becomes alkaline; this would cause a change in size, not of the nucleus or of the cortex, but of the whole mass of the concretion, and would necessarily fissure the latter through and through.

If the nucleus and cortex have not the same chemical composition, only one of the constituents may undergo chemical modification, and the fissuration may then involve only one. It is more difficult to explain why certain calculi break in irregular lines while others exhibit geometric regularity in their lines of cleavage. But when one considers the fragments of the latter one is led to see a resemblance with the mode of cleavage of certain crystals and to ask whether the amorphous or the crystalline structure of the salts composing any given stone does not explain the particular type of fragmentation of the one or of the other variety of stone. It is possible that crystallographic examination of these stones would solve this problem.

The stones that have been the least studied with reference to their fragmentation are those that occur within the kidney. These calculi, often coraliform, are phosphatic, and it is easy to understand how the least violence would cause them to rupture. Stones forming in the ureter or urethra lengthen as they grow, changing from the shape of the pit of an olive to that of a pit of a date and finally to a rodlike shape, through apposition of successive layers at the posterior or central extremity. When they are sufficiently developed they easily break along the lines of least resistance, by cleavage of the strata that have been deposited. Stones in the urethra are poorly protected, and it is not surprising that they break under trauma. The fragments then often become articulated so that they are termed articulated calculi. Vesical calculi show several kinds of fragmentation: (*a*) by cleavage, (*b*) by cracking and (*c*) by a combination of cleavage and cracking. The cracking may occur without any system of regularity or it may take place along lines of radiation oriented toward the center. According to some authors these radii start from the center, and when they reach the surface the fragments fall apart, dividing the calculus into regular halves, quarters and so forth. Chauvin found it possible in one case to produce the same type of regular cleavage by crushing whole stones in his hand. In the third type the cortex first separates from the nucleus, then these lines of radiation develop in the former while the nucleus remains entire. This type of fragmentation is seen most frequently in uric acid stones, and especially in hollow ones in which the nucleus has been resorbed. Oxalic calculi, the structure of which is, as a rule, regular and homogeneous, never undergo fragmentation.

CAUSES OF DEATH AFTER OPERATIONS ON THE URINARY TRACT

Hyman and Mencher [68] reported a study of the causes of death after operations on the urinary tract, reviewing 168 cases with 119 postmortem examinations.

There were 27 cases of renal calculus. The causes of death were: infection of the wound in 1 case, pyelonephritis in 12, peritonitis in 3, postoperative shock in 2, cardiac failure in 1, sepsis in 3, hemorrhage in 2, cerebral accident in 1, obstruction of the upper portion of the intestinal tract in 1 and general debility with decubitus and tabes in 1. In 11 of these 27 cases a bilateral involvement was found; in 9 cases the calculus was associated with pyonephrosis; in 3 solitary kidneys were present, and in 4 the patients were admitted in a state of uremia.

In the fifteen cases of tumor of the kidney the causes of death were: infection of the wound in 2 cases, pneumonia in 1, peritonitis in 1, shock in 3, pulmonary embolism in 2, hemorrhage in 3 and advanced carcinoma in 2. In 1 case death followed spinal anesthesia. One of the tumors was infected. In 11 cases nephrectomy was performed, and in 4 the surgical intervention was limited to exploratory operations, no further procedure being available. In 4 cases nephrectomy was performed by the transperitoneal route.

In 5 cases of renal tuberculosis death resulted from the following causes: renal infection in 1 case, tuberculous meningitis in 1, pulmonary embolism in 1, generalized tuberculosis in 1 and solitary kidney with advanced tuberculosis in 1. Two of these 5 patients were admitted in a state of uremia; 1 had bilateral tuberculosis; 2 had associated pyonephrosis, and 1 had calculus pyonephrosis. Two patients underwent nephrectomy; 2 nephrostomy, and 1, a transplantation of the ureter to the skin. In this group the disease itself was the prime cause of death.

In 10 cases of pyonephrosis death was attributable to the following causes: pulmonary embolism in 1 case, renal infection (original disease) in 5, sepsis in 3 and chronic glomerular nephritis in 1. Four patients were admitted in a state of uremia, and 1 entered with sepsis.

There were 10 cases of perinephric abscess, suppurative pyelonephritis and cortical abscess. The causes of death were as follows: peritonitis in 1 case, shock in 1, infection of the wound in 1, pulmonary embolism in 1, sepsis in 3 and renal infection in 3. Four of these patients were admitted in a state of uremia, and 2 with sepsis. In 2 cases nephrectomy was performed; in 5 only incision and drainage comprised the procedure, and in 3 nephrostomy was performed.

---

68. Hyman, A., and Mencher, W. H.: The Causes of Death After Urological Operations: A Study Based on One Hundred and Sixty-Eight Cases with One Hundred and Nineteen Autopsies, J. Urol. **33**:315 (March) 1935.

In the group of 72 deaths following operative procedures on the kidney for various conditions death was attributable to infection of some type in 40 cases (56.3 per cent). At least 30 patients died with uremic manifestations, 16 of them entering the hospital in a state of uremia. The principal factor in the causation of the uremia was infection.

There were 4 cases of calculus of the ureter; in these death was caused by infection of the wound in 1 case and by renal infection, cardiac disease and sepsis, respectively, in the other cases.

The causes of death in 25 cases of tumor of the bladder were as follows: infection of the wound plus other causes in 6 cases, pneumonia in 1, renal infection in 3, peritonitis in 2, shock in 2, cardiovascular disease in 2, pulmonary embolus in 1, sepsis in 4, undetermined cause in 2, thrombosis and gangrene involving the iliac artery in 1 and paralytic ileus in 1. In 5 cases of contracture of the neck of the bladder death was attributable to the following causes: peritonitis in 1 case, shock in 1, sepsis in 1 and renal infection in 2. All the patients in this group underwent suprapubic operations. The case of peritonitis was not attributable to any operative procedure but to rupture of an abscess of the spleen. Renal infection in 1 instance and pulmonary embolus in the other were the causes of death in the 2 cases of diverticulum of the bladder. In 2 cases of exstrophy of the bladder in 2 young children in whom the ureter was transplanted to the bowel death was caused by pneumonia in 1 and by intestinal obstruction at the site of implantation of the ureter in the other. The cause of death in all 4 cases of stone of the bladder was renal infection. In this group of 39 deaths following operation on the bladder, 25 patients (more than 64 per cent) died as a result of infection originating in the wound or in the urinary tract. The mortality from infection in the group of patients who had tumors was unusually high. The remaining causes of death are fairly evenly distributed among other complications.

There were 15 cases of adenoma of the prostate gland in which the first stage of suprapubic cystotomy was performed. The causes of death were as follows: pneumonia in 4 cases, infected kidney in 4, cardiac disease in 3, pulmonary embolus in 1, sepsis in 1, hemorrhage in 1 and phelgmonous colitis in 1. Six of these patients were admitted with acute urinary retention; 2 had vesical calculi, and 2 were admitted in a state of uremia. The causes of death in 13 cases of adenoma of the prostate gland, in which prostatectomy was performed in two stages, were as follows: infection of the wound in 1 case, pneumonia in 2, infection of a kidney in 3, cardiac disease in 2, sepsis in 2, hemorrhage in 1, diabetic acidosis in 1 and meningitis in 1. In 3 cases of adenoma

in which prostatectomy was performed in two stages, death resulted from the following causes: Infection of a kidney in 1 case, pulmonary embolus in 1 and sepsis in 1. In this group of operations on the prostate gland the general condition of the patient is important. Most of these patients are in the sixth or seventh decade of life and have, in addition to the prostatic disorder, coronary disease, myocarditis, generalized atherosclerosis or diabetes. Whereas in 45 per cent of the cases infection in one form or another was the important factor in the cause of death, the cardiovascular system and pneumonia accounted for an equal number of deaths. It is interesting to note that 15 patients died after undergoing ordinary suprapubic cystotomy. In a review of 294 cases of cystotomy and completed suprapubic prostatectomy, the mortality rate proved to be as follows: cystotomy only, 17 cases with 8 deaths; suprapubic prostatectomy in one stage, 44 cases with 3 deaths; suprapubic prostatectomy in two stages, 233 cases with 20 deaths. Although 11 patients died in an apparent state of uremia, the underlying factor was severe infection of the kidneys.

Primary causes of death in the entire series studied by Hyman and Mencher were as follows: renal infection in 45 cases, sepsis in 23, infection of the wound in 12, pneumonia in 11, peritonitis in 11, shock in 11, cardiovascular disease in 10, pulmonary embolus in 9, hemorrhage in 7 and cerebral conditions in 3—a total of 142 deaths. Contributing factors were these: uremia in 64 cases, renal infection in 19, sepsis in 2, infection of the wound in 17, pneumonia in 12, peritonitis in 6, shock in 3, cardiovascular disease in 4, pulmonary embolus in 3, hemorrhage in 3 and cerebral conditions in 1. Certain facts are emphasized by this study. There are complications which are unavoidable despite careful preoperative preparation, technical skill and assiduous postoperative care. Among these may be mentioned pneumonia, cardiovascular disease, cerebral factors and pulmonary embolism. Infection of the wound, to a certain extent, can be controlled. Excessive suturing of wounds in the presence of gross infection is to be avoided. Free drainage and protection of the perivesical and retrovesical region in all operations on the bladder is of prime importance. The necessity of protecting the peritoneum and immediately suturing all rents in that lining cannot be too strongly emphasized. Careful aseptic technic is essential to prevent introduction of infection during the irrigation of an organ through urethral and ureteral catheters and through suprapubic tubes or those used in nephrostomy. Control of hemostasis is obviously necessary. Another point to be taken into consideration is avoidance of excessive fulguration and irradiation of infected organs. Needless pyelographic and instrumental examinations should be avoided as much as possible, especially in cases of infection.

Braasch,[69] in discussing the causes of death after operations on the urinary tract expressed the belief that many of the deaths that resulted from infection following surgical procedures on the lower part of the urinary tract, such as prostatectomy and resection for tumor of the bladder, were caused by inadequate technic in drainage. Another cause of death is pulmonary embolism. This complication follows operations on the perineum, on the female pelvis and on the region of the prostate gland more frequently than operations on other regions. It is of interest to note that pulmonary embolism was observed in only 1 of approximately 1,000 cases of transurethral resections for obstruction by the prostate gland. It has been the experience of the Mayo Clinic that some of the newer substances employed for local and spinal anesthesia, and described as being harmless, have proved to be exceedingly toxic in some cases. Braasch mentioned a nonoperative death which resulted from the use of one of the newer "harmless" substitutes for cocaine, which was injected into the urethra. Death occurred instantaneously when injection was made into the urethra twenty-four hours after an attempt had been made to dilate a stricture. It is advisable to employ in the urethra after trauma only local anesthetics that are known to be innocuous. Renal insufficiency is undoubtedly an occasional cause of death. The various tests of renal function made prior to operation may give results that are normal or nearly so. In case of bilateral nephrolithiasis and stone in a solitary kidney, an elevated urea content of the blood of from 40 to 50 mg. per hundred cubic centimeters would not necessarily indicate renal insufficiency. However, in such cases, even after a simple operation, renal insufficiency occasionally develops, and death has occurred.

GASTRO-INTESTINAL MANIFESTATIONS OF UROLOGIC DISEASE

Portis and Grove[70] concluded, from a study of various obscure gastro-intestinal disorders, that urologic diseases are frequently the causes of referred symptoms. They suggested that the interrelationship of the nerve paths of the urinary and gastro-intestinal tracts may explain this curious relationship of symptoms. Women in whose urethras pathologic changes have taken place frequently show gastro-intestinal manifestations. Pressure phenomena and displacement are factors in a certain group of cases. Pathologic changes in the lower part of the urinary tract in male patients may cause symptoms, particularly in middle-aged patients. The gastro-enterologist and urologist should

69. Braasch, W. F., in discussion on Hyman and Mencher.[68]

70. Portis, S. A., and Grove, J. S.: The Gastro-Intestinal Manifestations of Urologic Disease, J. A. M. A. **104**:710 (March 2) 1935.

cooperate more closely to explain many obscure clinical pictures of abdominal distress which are the referred symptoms of primary urologic disease.

Brooks,[71] in discussing the gastro-intestinal manifestations of urologic disease, stated that nausea and vomiting are the most outstanding signs of renal disease. Pyelitis, renal stones, renal abscess or small cortical abscesses often cause these gastro-intestinal disturbances without markedly localizing symptoms pointing to the urinary tract as the affected part.

McKenna [72] stressed the frequency with which seminal vesiculitis causes referred pain at McBurney's point. Friedenwald emphasized the fact that stone in the pelvis of the kidney often produces symptoms resembling duodenal ulcer or cholecystitis, giving rise to pain several hours after meals, with signs of hyperacidity, or with reflex and colicky pain in the upper part of the abdomen. The true condition may remain obscure unless a thorough investigation is made. In a number of instances in which the patient previously had been under treatment for a considerable time for a supposed gastric cancer, subsequent examination indicated toxemia secondary to hypertrophy of the prostate gland as the cause of the nausea, vomiting and abdominal pain.

---

71. Brooks, Harlow, in discussion on Portis and Grove.[70]
72. McKenna, C. M., in discussion on Portis and Grove.[70]

Obituaries

E. STARR JUDD
1878–1935

Dr. E. Starr Judd passed away on Nov. 29, 1935, after a short illness. Death was caused by pneumonia. In his death surgery has lost one of its outstanding exponents. Possessed of rare surgical judgment, keen diagnostic sense and rare technical ability, he exhibited all the qualities of a master surgeon. He was born in Rochester, Minn., on July 11, 1878, and was graduated from the University of Minnesota School of Medicine in 1902. After serving an internship in St. Mary's Hospital at Rochester, he became an assistant of Dr. C. H. Mayo in 1903.

He successively passed through the grades of the hospital and medical school staffs and at the time of his death was surgeon to St. Mary's Hospital, professor of surgery in the graduate school of the University of Minnesota and head of a section in the division of surgery at the Mayo Clinic.

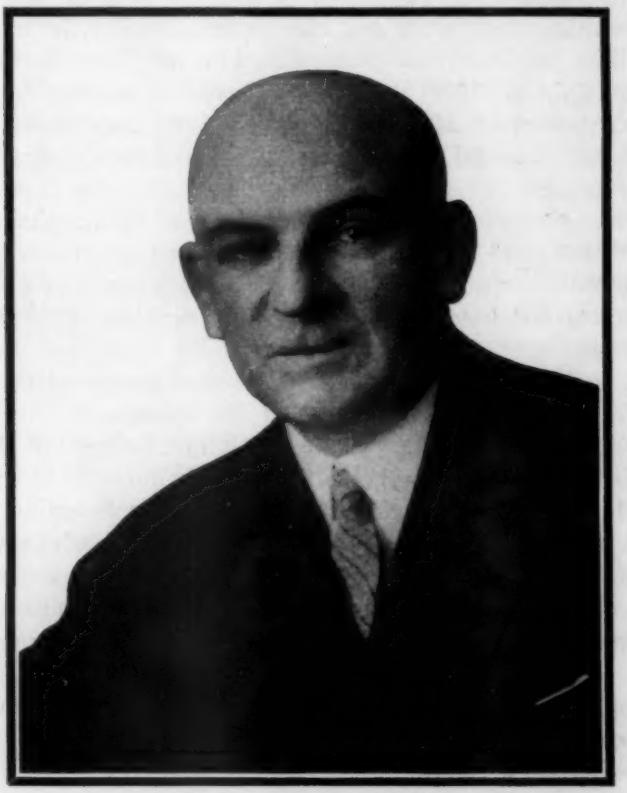
Early in his professional career he became interested in organized medicine. He served as president of the Minnesota State Medical Association and was secretary of the Section on Surgery of the American Medical Association in 1913-1916 and chairman of the section in 1918. He served as a member of the Council on Scientific Assembly from its beginning in 1915 until 1927. His sound advice was often sought in discussions concerning this assembly, which is an outstanding example of the value of demonstrations in the dissemination of the knowledge of newer things in medicine and the necessity of postgraduate teaching. He served as President of the American Medical Association during the year of 1931-1932. In this capacity he served with high distinction, ever mindful of the needs of the members of this organization and the objectives which they should hope to attain if they were to render the highest type of medical service.

Constant demands were made on him to appear before medical societies. Facts were presented so clearly by him, in such a simple but striking way, that he became a postgraduate teacher of great renown. He stood on solid ground surgically and was never greatly moved by surgical fads. Having the gift of critical ability, he dealt with fundamentals. Listeners knew that he spoke with the authority of experience and strove to sit at his feet.

He was a member of the American Surgical Association, the American College of Surgeons, the Minnesota Academy of Medicine, the Minnesota Pathological Society, the Western, Southern and Interurban surgical associations, the American Society of Clinical Surgery and the

Southern Minnesota Medical Association and an honorary and corresponding member of several foreign societies.

During the World War he was active as a teacher in the school for developing surgeons which was established at the Mayo Clinic, and from time to time since he has taken part in work designed to give those continuing in the medical corps of the United States Army a more intimate knowledge of diagnostic and operative procedures.



E. STARR JUDD
1878–1935

In 1921 Dr. Judd was made a member of the editorial board of the ARCHIVES OF SURGERY. His opinions have always been sought in determining the policy of this publication. He had a stabilizing influence, such as few possessed. Simple, almost shy, he was possessed of great wisdom; he could settle discussion by kind reason. He will be deeply missed by all who learned by association with him to know his greatness. His memory will be kept alive by the works which he has produced.

THE ARCHIVES OF SURGERY is published by the American Medical Association for the advancement of surgery and as a medium of publication for research, clinical observations and experimental work in surgery.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. Dean Lewis, Editor-in-Chief, Johns Hopkins Hospital, Baltimore, Md., or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF SURGERY, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF SURGERY. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones of illustrations will be supplied by the Association when the original illustrations warrant.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association. This requires, in order given: name of author, title of article and name of periodical, with volume, page, month — day of month if weekly — and year.

Matter appearing in the ARCHIVES OF SURGERY is covered by copyright, but, as a rule, no objection will be made to its reproduction in reputable medical journals, if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the ARCHIVES OF SURGERY or in any of the other publications issued by the Association will not be permitted.

Authors will receive one hundred reprints free; additional reprints may be obtained at cost.

The ARCHIVES OF SURGERY is issued monthly. The domestic annual subscription price (two volumes) is $8.00; Canadian, $8.40; foreign, $9.00, including postage. Single copies, 85 cents.

Checks, etc., should be made payable to the AMERICAN MEDICAL ASSOCIATION.

OTHER PERIODICAL PUBLICATIONS

of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (two volumes): Domestic, $7.00; Canadian, $8.50; foreign, $11.00. Single copies, 25 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced, original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $6.00. Single copies, 75 cents.

AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. It includes carefully prepared collective abstracts based on recent pediatric literature, abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $9.50. Single copies, 85 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $9.50. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and full abstracts of the literature on these two subjects, transactions of the important dermatologic societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A medium for the presentation of original articles on diseases of the ear, nose and throat, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original contributions in the field of pathology, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth-while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: Domestic, $12.00; Canadian, $14.00; foreign, $14.00.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn St. **CHICAGO**

CONTENTS

PAGE

Skeletal Changes in Malum Perforans Pedis. Jacob Kulowski, M.D., St. Joseph, Mo., and Robert Perlman, M.D., Brooklyn 1

Rôle of the Ileocecal Sphincter in Cases of Obstruction of the Large Bowel. Louis Sperling, M.D., Minneapolis 22

March Foot Associated with Undescribed Changes of the Internal Cuneiform and Metatarsal Bones. I. H. Maseritz, M.D., Baltimore 49

Acute Appendicitis and Associated Lesions: Some Observations on the Mortality. Rudolph N. Schullinger, M.D., New York 65

Postanesthetic Headache. P. W. Harrison, M.D., Muscat, Arabia 99

Neurofibromatosis, with Reference to Skeletal Changes, Compression Myelitis and Malignant Degeneration. Alexander Miller, M.D., Cincinnati 109

Transplantation of a Toe According to the Method of Nicoladoni: Report of a Case. Prof. M. I. Kuslik, Leningrad, U. S. S. R. 123

Congenital Anomalies of the Gallbladder: A Review of One Hundred and Forty-Eight Cases, with Report of a Double Gallbladder. Robert E. Gross, M.D., Boston. 131

A Review of Urologic Surgery (Concluded). Albert J. Scholl, M.D., Los Angeles; E. Starr Judd, M.D., Rochester, Minn.; Jean Verbrugge, M.D., Antwerp, Belgium; Alexander B. Hepler, M.D., Seattle; Robert Gutierrez, M.D., New York, and Vincent J. O'Conor, M.D., Chicago 163

Obituaries:

E. Starr Judd 191